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STUDY OF PHYSICS OF THE ANTRUM.*

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Upon being asked recently to discuss a paper on intra-antral air pressure incident to respiratory excursion and its effect on antral drainage, I was struck with the great paucity of knowledge on this particular subject. A few textbooks make some assertions but very little work has been done on the subject. Therefore, I thought it would be appropriate to go over some of the basic facts, fundamental to antrum drainage, and discuss the situation.

The maxillary antrum is a large bony cavity lined with a thin ciliated mucous membrane with one or more openings half way up the inner side of the cavity. Drainage is said to be affected by aspiration, position of the head through the force of gravity, the action of the cilia and to some extent by cohesion of secretion and capillary attraction between the walls and any fluid therein. We will discuss the question of air pressure at length and dismiss the other physiological factors with brief comments. Investigation has shown that the cilia wave toward the ostium. This work has been done by Schaeffer

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and others, using carbon particles which are seen to gradually move toward the ostium. In the nasal cavity they have been shown to carry particles toward the nasopharynx. Very ingenious apparatus has been constructed to measure the amount of air passing through the nose and the effect of changes of respiration on the air and the pressure of the same, but I could find no account, except the work of Dr. McMurray[1], of any effort to measure the intra-antral air pressure. W. Undritz[2], working in Noyatschek's Clinic in Leningrad, has called attention to the fact that the act of respiration through the nose may call forth reflexes in the muscles of respiration and the lung. It has occurred to me that it is possible there may be a rhythm between inspiration and the movement of the cilia. This has not, to my knowledge, been studied. Undritz constructed what he called a rhinoanemometer to measure the velocity of the current of air. From the velocity he calculated the pressure by a mathematical formula as follows:

P = pressure.
V = volume passing through in unit of time.
T = time.
K = constant coefficient.
W = resistance.

"The greater the P, the greater the V (direct dependence). The stronger the resistance met in the nasal cavity, the smaller the V (inverted dependence)."

$$V=K\ \frac{P.T}{W}$$

"The capacity of the nose is inversely proportional to the resistance. The smaller the resistance, the better the passage of air."

$$D=\frac{1}{W}$$

The rhinoanemometer is a tube in which a small wheel is inserted which deviates with the air passing through, giving us a reading directly on a scale of the number of cubic centimeters of air expired a second.

He found that an adult draws 500 c.c. at every quiet respiration, or at 18 per minute we have 9 litres per minute. This tidal air is only a part of the vital capacity of the lungs. Glotzel, and later Brunning have used polished metalic plates to study the relative capacity of the nostrils and the effect of inspiration and expiration. Brunning tested each nostril separately, both for inspiration and expiration, and then tested them together. He found that the normal time for expiration with both nostrils open is one second, but when

he tested each one separately he found it was two seconds. Hochrein[3] has done a great deal of work with the pneumotachygraph. He and Fleisch have carried on a rapid fire of argument over the relative merits of their machines. These were developed essentially, however, for study of the lung but, incidentally, they have gathered valuable data about the nose.

With the idea of studying the pressure in the nose on inspiration and expiration and checking some of the figures given by the authors quoted, I examined about twenty patients. I used a water U-manometer for gentle respiration and a mercury U-manometer for forcible respiration. I carried out this work by taking a small glass cannula and passing it into the vestibule at times and at other times further back toward the choanae. I found that in gentle respiration we have a negative pressure on inspiration of from —4 to —16 m.m. of water and on expiration plus pressure of 1 to +10 m.m. of water. I then measured the pressure in the antrum by introducing a cannula through the lateral wall of the nose under the lower turbinate and found it the same in the antrum as in the nasal fossa. In antra in which I found the pressure differed from the pressure in the nasal fossa, I found that there was pus in the antra or that our tubes were blocked with a droplet of water or, as in the instance of introducing an extra cannula into the antrum under the middle turbinate, to simulate an accessory ostium, as advised by McMurray, we were allowing atmospheric air pressure to overcome the pressure in the antrum. The study of these pressures led us to the question of velocity head. You will see at once that if we insert a straight cannula and find that we have a negative reading for inspiration, we are measuring the pull of the air as it rushes past the orifice of the cannula which has been placed in the fossa along the floor in the same direction as the current which we are measuring. On expiration the air is then rushing in the opposite direction to the cannula and tends to rush into the orifice, thus giving us a positive pressure. You will at once see that if we reversed this cannula and took our measurements again the results would read differently. The real factor which we wish to observe is what is called the velocity head. This may be measured by a Pitot tube, which would take both readings and give the resultant. To simulate this I made some experiments, as follows: I established that the pressure was the same in each nostril in a given sense. I then put a cannula, as usual, in one nostril and bent a cannula on itself and introduced it into the other nostril. Thus in each excursion of inspiration and expiration I would obtain one reading with the air going toward the orifice in a cannula and an-

other reading with the air rushing by the orifice in a cannula. These experiments were very crude and I feel that they could be repeated with great benefit in a more careful manner. I feel that our statistics so crudely gathered are not accurate. I found that the two forces partly equalized each other in the manometer and that the velocity head on quiet respiration was —6 m. of water for inspiration and +2 m. of water for expiration. I then measured the pressure in the nasal cavity and in the antrum on blowing the nose and on forcibly sucking air through the nose, as in clearing it. I could not notice any difference of pressure between the antrum and the nasal cavity. I find that on blowing the nose we can get a pressure of from 60 to 120 m.m. of Hg. or a water pressure of 1088 to 1632 m.m. of H_2O, and upon suction we get a negative pressure of 30 to 100 m.m. of Hg., or 408 to 1360 m.m. of H_2O. With the manometer in the vestibule, I did not take the measurements with the mercury manometer simultaneously in the nasal cavity and in the antrum. Practical engineers in the production of vacuum pumps tell us that they can usually produce a negative pressure of one-fourth the positive pressure given them to work with.

I think that this work should be done over again, using simultaneous readings with manometers that would record their pressures on a photographic film and which could be compared at leisure with each other and with the various phases of respiration. Such a manometer has been devised by Dr. R. M. Lewis and used in measuring spinal fluid pressures.

When we come to look into the question of pressure in the antrum produced by respiration we find that a number of problems present themselves. If the negative pressure is altogether due to aspiration, what effect will respiration in the opposite direction have? For instance, if the antrum is aspirated upon blowing the nose, what happens when we draw the air in? Is there simply a replacement by equalization of pressure through the ostium, or is the air actually blown into the ostium on inspiration? Another question that could be studied is whether there is aspiration during both phases of respiration. In order to account for negative pressure inside the antrum that is greater than the negative pressure in the nose, we must conclude either that the pressure is temporary, as during inspiration or expiration, or that it is present during a period longer than the respiratory excursion. It would seem that the latter theory would demand some valve mechanism which would prevent pressure in the nasal cavity equalizing pressure in the antrum, when the direction of the current of air is reversed during respiration. If there is such

a normal mechanism for building up a greater negative pressure in the antrum than in the nasal cavity, any extra hole or holes as an accessory ostium in the antrum would, in my opinion, simply tend to equalize the pressure between the two cavities, but by their very size improve drainage, the larger the better. If there is no valve-like action, the negative pressure inside the antrum could never exceed the negative pressure in the nasal cavity, except merely during one phase of respiration. As soon as the reverse phase begins, the pressure in the antrum changes. I think that the effect of respiration on drainage of the antrum through the normal ostium may be explained by the theorem of Bernoulli, which is that when water flowing through a pipe meets a constriction, the lateral pressure diminishes in proportion to the velocity of flow. It may be demonstrated by the following illustration, which shows that the manometer erected at

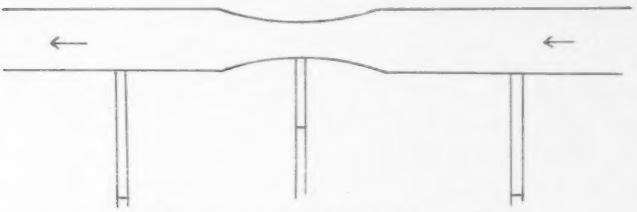

Fig. 1. Study of physics of the antrum.

the point of constriction, shows less pressure than the manometers erected at points where the pipe is of full diameter. One may see, that in order to maintain an equal flow in the pipe, the fluid must rush by the constricted point much more rapidly than other points. At the constricted point, so to speak, it does not have time to build up any lateral pressure. This fact, that lateral pressure lessens with speed of the current, accounts for some interesting phenomena. For instance, it is well known that ocean vessels must approach each other with the greatest care. The reason is that, according to the theorem of Bernoulli, a current being present going through the constricted area between the vessels, we will have a lessening of the lateral pressure on the sides toward each other and a greater pressure on the sides away from each other, thus tending to force the ships together (see Fig. 2.)

Another case demonstrating this principle is to take a flat disc through the center of which we lead a compressed air current to a small orifice. A little celluloid ball is placed several inches above

the orifice and the force of the current will blow the ball into the air, but when we place the ball directly over the orifice on the disc the air rushing out with a given speed around the adjacent hemisphere results in a lower lateral pressure at this point, the result being that the atmospheric pressure above will hold the ball to the disc, thus with considerable pressure blowing out through the orifice, the ball is held tightly to the disc. It is this same theorem of Bernoulli's and the physical principles which it sets forth that apply to the drainage of the antrum. I believe that the curling of the turbinate directs the air into the infundibulum during expiration and has the effect when rushing through the narrow space, and by the ostium, of lowering the lateral pressure at this point, compared with the nasal cavity as a whole, thus favoring a certain amount of suction in the antrum.

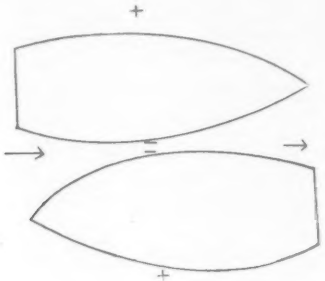

Fig. 2.

But, if one or more accessory ostia are present, negative pressure of inspiration will all the more easily be transmitted to the antrum.

I think that the position of the infundibulum slanting from the above downward and before backward, would seem to favor aspiration during expiration. This same position would favor blowing into the antrum on inspiration. I think that this is what takes place, unless the force of expiration, as in blowing the nose, is out of all proportion to the ability of the ostium to convey air into the antrum. In other words, on gentle expiration air goes into the antrum; on forcibly blowing the nose, aspiration comes into effect, but we have not proved it by our experiments.

Another way in which the antrum may be drained when secretion is present, is by the entrapping of air similar in a degree to the principle of exhaustion used in mercury pumps. The mercury vapor passing an orifice, carries some of the air with it. When secretion

is presented at the ostium by the wave motion of the cilia, aspiration may lessen the air in the antrum, but when the cohesion of the secretion with the mucous membrane surrounding the ostium and the mass of the pus are sufficient to withstand the pressure built up, it is conceivable that secretion at the ostium might snap back into place and hold a certain degree of negative pressure in the antrum, or the air rushing past would entrain the small globule of secretion and, another taking its place at the ostium, the air between the two globules would be entrapped, causing some minus pressure in the antrum and aiding drainage. In all these instances, however, I think accessory ostia would improve drainage rather than impair it.

When the antrum becomes full of secretion, the mechanism of drainage may not be the same as in ordinary respiration in a normal case. When the nose is forcibly blown and air directed against a swollen turbinate it would appear from the position of the infundibulum that the air might in some instances be directed into the antrum, causing some eddy currents and forcing some of the pus out through the same opening. If such a mechanism takes place, the presence of accessory ostia would be an advantage, in that the pus could more easily go out the second opening. However, our tests with the manometer in the antrum showed some negative pressure upon sucking and positive pressure when the nose was forcily blown. We noticed that whenever our pipette became obstructed, the pulse beat could at once be read from the manometer.

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THE USE OF A PERIOSTEAL FLAP WITH SKIN GRAFT IN RADICAL MASTOID SURGERY.

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Radical mastoid surgery is looked upon with fear and trepidation by many members of the medical profession. There is some foundation for this view. The mortality is relatively high, complications and sequellae are not uncommon. In this, as in other types of chronic pathology, the social and economic factor plays an important role. The anatomy of the temporal bone, especially of the petrosal pyramid, is of tremendous importance in the ultimate success or failure. It is probably the most important single factor. The excellent researches of Kopetsky[1], Eagleton[2], Shambaugh[3], Wittmaack[4], Portmann[5] and others have clearly demonstrated the variability of its cellular structures. The close proximity of the important functional and vital structures is of paramount importance. Roentgen studies[6], though a great aid, are still of real value only when properly interpreted in the presence of positive findings. Time and research will no doubt increase the value and importance of Roentgen studies.

The anatomy of the tympanic cavity, aditus and antrum, is such that mastoid surgery in a great measure resolves itself into a problem of drainage. This is true of whatever method may be used: simple, radical, modified as advocated by Mr. Heath[7], of London, or intra-aural surgery recently revived and popularized by the Doctors[8] Tobey of Boston. Drainage, one of the great fundamentals of surgery, must not be forgotten in this as in all other types of surgery, where infection is or has been a factor. Accordingly extension of pathological processes is prevented or at least an attempt is made at prevention. The importance of this is evident when the proximity of the physiological and anatomical structures is recalled. Upon these academic truths the otolaryngologist is compelled to work in millimeter distances. Though the technical difficulties are much greater than in other types of surgery, the fundamental principles of surgery remain: the anatomy, physiology, bacteriology, and pathology of the part involved and their relations to the entire human mechanism.

The indications for radical mastoid surgery, relative or absolute, are so well known to students of the subject, that it seems superfluous to even call attention to them. Suffice to say that a positive

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indication is an intracranial complication in a chronic discharging ear. Relative indications depend in a measure upon the training, association and experience of the surgeon. The prevalent opinion is that any chronic purulent discharging ear after a period of three months of proper treatment, either medical or surgical, becomes a potential case for radical mastoid surgery. This is particularly true of peripheral perforations with bony necrosis, also evidence of facial nerve disturbances and, in most cases, of cholesteatoma. A knowledge of the pathological process and one's ability to analyze it and also the clinical findings applies here as in any other type of surgery. A strictly middle ear infection, with no bony necrosis, with the presence of a tubal involvement, even though the discharge was persistent and

Fig. 1. Anatomical demonstration of post auricular incision, periosteum intact and fleshy portion of external auditory canal retracted forward.

of long standing, would not be an indication in my opinion. My message is that the relative indication depends upon the pathology present, one's ability to analyze the condition and the effect upon the patient as to the immediate and remote mortality, morbidity and disturbance in the physiology of adjacent and distant structures.

It is unfortunate that the social and economic conditions of a patient have a great bearing upon his consulting a practitioner as well as upon his accepting his advice. This is especially true of patients with an indication and who have been properly advised in regard to mastoid surgery. Chronic surgical pathology may be present for years and continue for many more without serious trouble. The public is well aware of this, and the fact is capitalized by various cults

many times to the detriment of the patient. So procrastination is the result. Add to this the long period of convalescence, the cost in time and money, and can we wonder why thousands of individuals afflicted with chronic discharging ears procrastinate? Yet many of these are capable of securing good surgical results with avoidance of future serious complications.

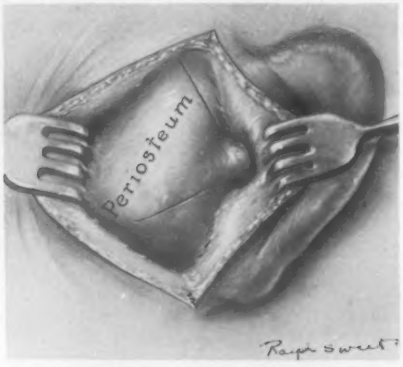

Fig. 2. Photograph of periosteal incision.

Fig. 3. Photograph of separated and retracted periosteal flap with radical mastoid cavity ready for return of flap.

Numerous techniques and modifications of the original radical procedure have been advocated, some with the purpose of a better surgical result, others with the object of lessening the cost in time and money. Most of the modern literature of radical mastoid surgery deals indirectly with this economic phase. This is particularly noticeable in the various methods of skin grafting the cavity to obtain a more rapid epithelization[9, 17].

A few years ago Dr. D. Campbell Smyth[14], of the Massachusetts Eye and Ear Infirmary, advised a method which, with some modifications, I have found highly satisfactory. It certainly lessens the period of convalescence. Until adopting this method I had been doing the radical procedure with or without the use of a skin graft.

Fig. 4. Photograph of mastoid cavity with periosteal flap and skin graft superimposed.

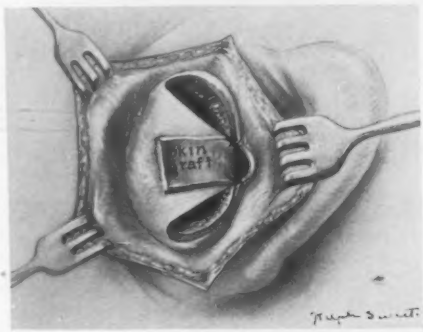

Fig. 5. Photograph of mastoid cavity with periosteal flap and skin graft in position.

Briefly, the technique is as follows: After proper preparation of the external auditory canal and operative field, the usual postauricular incision is made down to, but not including, the periosteum. The fleshy portion of the external auditory canal is retracted forward (see Fig. 1). An incision is made into the external canal at the juncture of bony and cartilaginous wall sufficiently large to view the bony external auditory canal. With a small right angled knife the

periosteum of the posterior bony auditory canal is cut near to the tympanic ring. Two linear incisions made through the periosteum of the inferior and superior canal wall are carried externally from the extremities of the original transverse incision. At the area where the posterior linear incisions meet the periosteum of the external mastoid surface they are carried upward and downward in a fan shaped direction (see Fig. 2). The size and extent of this fan shaped area depends upon the type of case and judgment of the operator. This total area of periosteum covered by modified skin of the external bony canal (which is easily accomplished by the use of an ordinary submucous elevator) is dissected free from the underlying bone, both of the external canal and mastoid process. It is then retracted backward

Fig. 6. Photograph of completed operation, with wick in ambrine to facilitate removal via the external auditory meatus.

and covered with a moist sponge (see Fig. 3). At this stage the radical mastoid operation is continued and completed as the operator sees fit. After the completion of the bone exenteration and removal of pathology, the cavity is dried (see Fig. 3); then the periosteal flap is placed in position and covered with a skin graft, external to the tongue shaped area representing the periosteum and skin of the external auditory canal (see Figs. 4 and 5). In this way the cavity is partially lined with periosteum with skin superimposed. The grafts can be applied directly to the periosteal flap under traction before placing it in the cavity or in situ. It is immaterial. Then any of the various types of plastic flaps from the fleshy portion of the external canal is made and anchored to the upper and lower portion of the

incision. I have found the modified Koerner to be very satisfactory. The upper and lower angles are anchored to the skin incision with mattress sutures. The posterior wound is closed in the usual manner. I prefer the use of wax substances such as paraffine or ambrine to packing with gauze (see Fig. 6). My results are more satisfactory, the convalescence is shortened and the pain of postoperative use of packing is eliminated.

This technique differs from that advocated by Dr. Smyth in that the cutting in the external auditory canal is through the mastoid incision. I do not carry the incision as far internally as Dr. Smyth advocates which is to the promontory. The skin grafts placed on the periosteal flap in the excavated cavity are utilized by me with the purpose of more rapid epithelization. I have also found that the wax inlay has the same advantage. In my method the tip of the skin periosteal flap does not enter the middle ear cavity, neither does it in the mastoid advocated by Dr. Smyth. To overcome this I use a skin graft as he describes for the middle ear.

Recently I did a double radical mastoid on a patient—each side at a different time, of course. The indication was chronic suppuration with extension into the petrosal angle. These findings were corroborated by Roentgen studies by Dr. Monica Donovan of San Francisco and found to be present at the time of operation. The total time of immediate convalescence on the left side was 44 days, 16 in the hospital and 28 in the office. The patient was then dismissed with advice to return at monthly periods for cleaning and observing the canal. A radical mastoidectomy was performed on the right side two months after that on the left side. The periosteal flap and skin graft was used. The hospital stay was shortened to nine days, the patient's immediate convalescence was complete in 26 days. I could recount other cases studied in a similar manner with comparatively the same results. Every case is a law unto itself, still, I am convinced from a limited experience—and, after all, most of our experiences are limited when we analyze radical mastoid surgery—that the method I present lessens the time of convalescence 30 to 40 per cent, no small factor in the social and economic life of these patients.

In the cases where the dura, sinus or labyrinthine structures are exposed this method is not advised. These constitute a small minority, still they should always be kept in mind.

In presenting this method, due credit is given Dr. Smyth[14] for his originality in devising the periosteal flap. My addition to the technique is the utilization of a skin graft and a slightly modified periosteal incision from that first advocated by Dr. Smyth. I commend it for trial in indicated cases.

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GENERAL AND OTOLOGICAL ASPECTS OF CHOLESTERIN METABOLISM.*

Dr. Maxwell Fineberg, St. Louis.

Much research has been done in the attempt to prove a common relationship between arteriosclerosis, essential hypertension, chronic nephritis due to contracted kidney, apoplexy and chronic progressive internal ear deafness. The prevalent opinion until 1921 was that a primary arteriosclerosis formed the basic etiological factor. This interdependence of diseases formed a vicious circle, which was not broken until 1921, when Volhard and von Bergmann[1] advanced their theory that essential hypertension should be considered a disease sui generis. Their theory has gained much universal credence in the last nine years, and it must be agreed that there is sufficient basis for their theory to better explain those clinical pictures which up to that time had to be explained by a vicious circle.

The work of Volhard and von Bergmann stimulated research to ascertain what might be the underlying cause of this new but very common disease. Verse[2] in 1925 reported that he believed metabolic changes were primarily responsible for essential hypertension and that the metalobolic changes might be the connecting link in the relationship between essential hypertension and those diseases which, prior to Volhard and von Bergmann's theory, had been attributed to primary arteriosclerosis.

Autopsy material was then examined more carefully for the presence or absence of arteriosclerosis in cases of this group of diseases. The reports were conflicting, in that where the clinical history was suggestive of arteriosclerosis, the pathologists reported that there was little or no evidence of it. They frequently found the most marked evidence of arteriosclerosis in those cases which had no clinical mention of the condition.

Rosenblath[3] had previously shown that apoplexy should not be considered as due to gross hemorrhage in the brain but rather as due to numerous pin-point aneurysmal diapedesic bleedings. Westphal and Bär[4] elaborated on the work of Rosenblath and came to the conclusion that arteriosclerosis has nothing to do with these diapedesic bleedings. They claim that both of these conditions and hypertension as well are the direct result of spastic vascular changes.

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But what is it that causes these spastic vascular changes? What is it that directly or indirectly causes arteriosclerosis, hypertension, apoplexy and spastic vascular changes? Is there some factor which may be the connecting etiological link in these conditions?

The writer believes that there is a definite connecting link which may be found in the cholesterin metabolism in cases of arteriosclerosis, essential hypertension, apoplexy, chronic nephritis due to contracted kidney and progressive internal ear deafness. There is a very clearly marked hypercholesterinemia in all these conditions and it is the opinion of the writer that the hypercholesterinemia is not at all coincidental, but that it plays no small part in the etiology of all these conditions.

There is quite definitely some close relationship between arteriosclerosis and chronic progressive ear deafness. The deafness was and is sometimes still considered due to an arteriosclerosis of internal auditory vessels. Alexander[5] in 1902 actually demonstrated the sclerotic changes and since then it has been taken for granted that arteriosclerosis produces an internal ear deafness. This, however, does not explain those cases which do not show any generalized arteriosclerotic changes; nor does it explain why so few specimens of sclerosis are reported in the literature.

There are numerous theories cited for the cause of chronic progressive internal ear deafness. Boenninghaus[6] believes that senile deafness and presbyacusis are caused by a degenerative process in the acoustic nerve. He says that one frequently finds a nerve impairment of hearing in premature arteriosclerosis which is clinically manifested by shortened bone conduction and a lowering of the upper tone limit. On the other hand, in cases of presbyacusis one frequently finds an irritative condition of the acoustic nerve as manifested by head noises and dizziness. Thus Boenninghaus concludes that presbyacusis has to do with an acoustic neuritis which may often be caused by the same factors which cause an early arteriosclerosis.

Zwaardemaker[7] was among the earliest to investigate senile deafness. He concluded that the lowering of the upper tone limit was a normal physiological phenomenon which began in the third decade and then gradually increased.

Sporleder[8] thought that severe and even middle grade deafness must be produced by factors other than age. O. Mayer[9] suggests that "marasmus senilis" may be responsible for changes which are beyond physiological limits of normal. Lederer[10] agrees with O. Mayer in substance but says it is impossible to estimate the age at onset of these physiological phenomena. Stein[11] lays much stress

on arteriosclerosis as being the cause of senile deafness and he claims that there is an accompanying local anemia which accounts for head noises.

From these few references it will be readily agreed that there is still some important link missing in the chain connecting arteriosclerosis, hypertension and senile progressive deafness. In delving further into this problem it was thought that the examination of apoplectic patients might shed some light on the relationship. Working in conjunction with Privat-Docent Josef Berberich, of Frankfurt am Main, the writer examined fifty-eight cases of apoplexy with a view to ascertaining whether there were any defect in hearing, and if there were, whether it was associated with the apoplexy. The vast majority of our cases had a very definite hypertension. Much stress is laid on this accompanying hypertension because it is believed that the internal ear changes found were related more or less closely to this phenomenon. We[12] found rather uniformly in fifty-eight cases of clearly proven apoplexy that there was an internal ear deafness of varying intensity. Subjectively, the patients complained more frequently of head noises than of impairment of hearing. Because of the constant presence of hypertension in our cases and the frequency with which we met the symptom of head noises, we were led to make the following statement: a patient who came to the doctor complaining of head noises and who showed a hypertension should be considered a possible eventual apoplectic.

The deafness in these cases was bilaterally equal and had no relation to the age of the patient or to the apoplectic insult. What might cause such an internal ear deafness? The conditions which come into consideration are occupational deafness, intoxication, chronic poisoning, lues, diabetes, nephritis and atypical forms of otosclerosis. Our cases were institutional and therefore, by careful examination, we were able to rule out all of the mentioned possibilities. Nevertheless, we remained with an internal ear deafness which could not be satisfactorily explained. We therefore deduced that the deafness must be associated in some way with the accompanying hypertension.

In discussing the work of Rosenblath, Westphal and Bär, Verse and others on the question of hypertension, the opinion was expressed that hypertension and hypercholesterinemia were closely related. The theory is now advanced that hypercholesterinemia may be the missing etiological link in the chain formed by essential hypertension, arteriosclerosis, apoplexy and chronic progressive senile deafness.

There is no doubt that these conditions are separate entities and yet have something in common. If it can be definitely proven that

an increased cholesterin metabolism plays some role in the etiology of these various conditions, then new avenues of approach will have to be opened to study their cause and treatment. The purpose of this article is to show that an increased cholesterin content of the blood produces changes which are common in the various conditions under discussion. Arteriosclerosis, hypertension, apoplexy and chronic nephritis due to contracted kidney may either individually or collectively be closely associated with chronic progressive internal ear deafness. For this reason a search was made of the literature for histological information in chronic progressive internal ear deafness. Politzer and Morpurgo[13] reported atheromatous changes in the arteria auditiva interna in a case of arteriosclerosis. At a later date Politzer[14] in his textbook states that atrophy of the acoustic nerve may arise following circulatory changes. Alexander[5] described the histological changes he found in a case of arteriosclerosis. Manassa[15] described the histological picture of chronic progressive internal ear disease and pointed out various locations where the atrophy and degeneration were most marked. He blamed arteriosclerosis for causing these changes.

Kashiwabara[16] in a case of apoplexy saw scattered hemorrhages in the internal ear. He could not demonstrate any breaks in the blood vessels so he termed the hemorrhages diapedesic bleedings. In later years Rosenblath and Westphal and Bär proved that apoplectic hemorrhages into the brain were also of a diapedesic character. This finding of Kashiwabara may prove of great interest in the study of "Meniere's symptom-complex."

Stein[17], Jaehne[18], O. Mayer[9], Wittmaack[19], Stuerer[20] and others report numerous cases which show similar changes. They advance either arteriosclerosis or hypertension as the basic cause.

Thus far, it has been possible to link up the various conditions under discussion. Chronic progressive internal ear deafness seems to be an outstanding feature of them all. The next problem is to connect these conditions to what is advanced in this paper as a possible etiological factor, namely, a hypercholesterinemia.

At this point a brief discussion of cholesterin metabolism is in order. The cholesterin content of normal blood serum is fairly constant, varying between 12 and 18 m.g. per 100 c.c.m. of blood. A hypercholesterinemia may be traceable to the following factors:

1. To nutritional increase from ingested food. The increase, however, is not constant and is of a transient nature.

2. To cell destruction. Due to this, the cholesterin depots become overloaded and a circulatory surplus ensues. This surplus may then

form a synthesis with enterogenous resorbed bile. In either event a higher serum cholesterin results. This theory was advanced by Hueck[21].

3. To endocrine disturbances, especially those of the adrenal gland.

4. To abnormal changes in the sex glands. Castration increases the cholesterin content of blood serum. It is also raised in the latter half of pregnancy. Eufinger and Bader[22] were able to show that this increase remained some time in postpartem women who were of the nervous type.

5. To changes in the thyroid gland. Cretins show an increased blood serum cholesterin. This is in keeping with the experimental evidence of Leupold[23], who fed thyroid to animals.

6. To constitutional factors. To this group belong the people whom Westphal terms "typus apoplecticus" because they have a hypertension, an increased blood serum cholesterin and a lack of resistance to inflammatory processes.

7. To hitherto unexplained factors of unknown origin.

Westphal[4] propounds an interesting theory to explain how cholesterin maintains smooth muscle tone. He experimentally shows how a strip of arteriole contracts more vigorously in a solution containing cholesterin. Westphal then examined patients with hypertension and found that 89 per cent of them had a hypercholesterinemia. From his investigations he formulated the following conclusions:

1. Hypercholesterinemia and hypertension are closely associated.

2. An increased cholesterin content is found in young patients suffering from increased blood pressure, so that,

3. Arteriosclerotic contracted kidney cannot be a cause, but more probably is a sequela of the disease.

From these deductions, an explanation may be found for numerous transient conditions, *e. g.*, Meniere's, vague attacks of dizziness, etc.

It is not to be wondered at that definite histological changes in temporal bones of beginning senile deafness have hitherto not been demonstrated. The early symptoms depend on functional changes in the labyrinthian vessels. Only when the disease has been of long standing is it possible to histologically show a continued contractile condition of the vessels together with a cholesterin deposit. At this stage the changes are similar to those found in the kidney, spleen and pancreas.

In the work previously referred to[12], we reported on the histological findings in nineteen cases, in six of which we had been able to do hearing tests shortly before death. All six cases showed an internal ear involvement of varying degree. The nineteen cases were

comprised of seven apoplectics, six diabetics, two arteriosclerotics, one pyemic and two uremics following chronic nephritis, and one case of frontal sinus empyema. The ages of these patients varied between 49 and 85 years.

The temporal bones were prepared in accordance with Wittmaack's method and then embedded in gelatin. Serial sections were cut and then stained with scarlet red and hematoxylin.

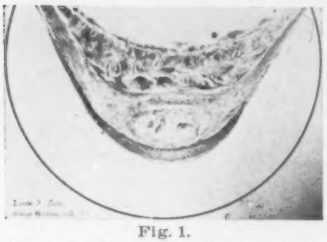

Fig. 1.

Fig. 2.

Fig. 1. Arcus lipoides myringes and hammer handle in 66-year-old diabetic.

Fig. 2. Otitis media in 28-year-old woman with nephritis and hypertension. The inflamed mucous membrane has undergone fatty changes.

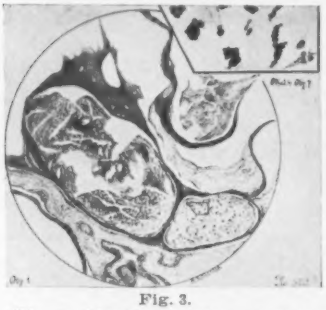

Fig. 3.

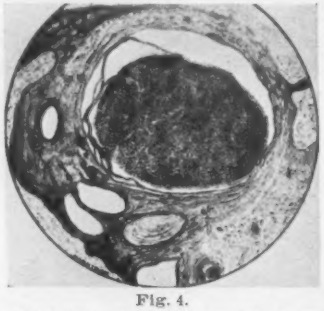

Fig. 4.

Fig. 3. Lipoid changes in a case of mastoiditis. Cellular exudate shown in small upper section.

Fig. 4. Bony cells of facial canal show ring-form storehouse of fat and lipoids.

At this point it should be explained that various terms have been used in the designation of identical histopathological changes. Thus, in this article, the terms lipoid deposit, fatty lipoid deposition, fatty deposit, fatty or lipoid changes are all used to designate different degrees of the same pathological entity.

The histological changes found in all nineteen cases were similar, so we shall only point out individual factors of interest in the group.

A typical arcus lipoides myringis (see Fig. 1) was seen in nearly every case. The lipoid deposits in the tympanic cavity are seen only in the area of the tube opening. One never sees lipoid deposits in the submucous layers except where there are coexisting inflammatory changes. The inflamed mucous membrane has undergone some fatty change (see Fig. 2). The lipoid deposit is seen only in the newly formed inflammatory tissue and cannot be seen in the intact normal tissue. The ossicles and secondary tympanic membrane do not show any characteristic lipoid changes.

The lining of the mastoid cells normally shows no fat except for normal fatty medullary tissue. However, lipoid changes occur in our cases where an otitis was coexistent (see Fig. 3). One of our cases died of meningitis following labyrinthotomy and the newly formed granulation tissue showed the typical lipoid deposits.

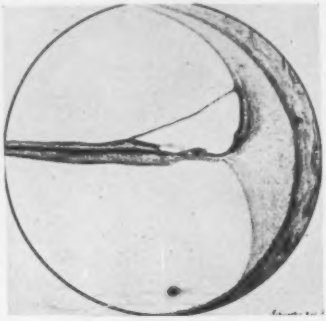

Fig. 5.

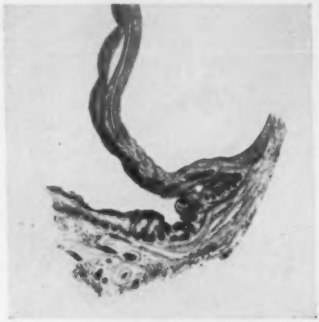

Fig. 6.

Fig. 5. Reissner's membrane shows fine fatty droplet formation in the elastic fibres. The stria vascularis and capillaries show lipoid deposits.
Fig. 6. Experimental arcus lipoides in cholesterin-fed rabbit.

Our cases with coexistent inflammatory changes show without question of doubt, that when the blood contains an increased amount of cholesterin, it will be deposited in newly formed connective tissue and cellular elements. This confirms the belief that the process has to do with an equalization or stabilization of the vital storehouses of the reticuloendothelial apparatuses.

The chorda tympani and facial nerve were unchanged. The bony cells of the osseous facial canal showed a definite ring-form storehouse of fat and lipoids (see Fig. 4).

The cochlea showed in every case, but more particularly in the diabetics, some lipoid deposits in the stria vascularis and its epithelium. The capillaries are partially filled with fat and the pigment of the epithelium is no longer to be recognized (see Fig. 5).

The prominent vessel between the stria vascularis and the external spiral sulcus shows in particularly nice fashion what happens in a hypercholesterinemia. The wall is covered by fat, exactly as we find it in the kidney, spleen and other organs.

The cells of Claudius show changes only in the advanced diabetics. The cells of Hense and Deiter show lipoid deposits which are more marked in the cells nearest the lumen of the ductus cochlearis.

The membrana tectoria shows no changes. The internal spiral sulcus, the membrana basilaris, the ligamentum spirale, the stria vascularis and Reissner's membrane show characteristic lipoid deposits.

The vestibule and semicircular canals show no histological changes.

In summing up our histological findings, we may say that our nine-

Fig. 7.

Fig. 8.

Fig. 7. Experimental lipoid deposit about annular ligament of stapes.
Fig. 8. Cochlea in cholesterin-fed rabbit. Lipoid deposit is marked in the stria vascularis, ligamentum spirale and in the cells of Hense, Claudius and Deiter.

teen cases of apoplexy, diabetes, hypertension and nephritis, all showed a typical lipoid infiltration in the drum membrane, in the ossicular chain joints and in the inflamed mucous membrane of the middle ear and mastoid process. All of our cases show a typical lipoid infiltration in the isolated elements of the ductus cochlearis, of the ligamentum spirale and of the stria vascularis. The small arteries and precapillaries show changes which are similar to arteriosclerosis. Thus we conclude that an increased cholesterin content in the blood produces a lipoid change in the peripheral auditory end organ.

It was stated earlier that the object of this paper was to link up hypercholesterinemia with arteriosclerosis, apoplexy, hypertension, chronic progressive internal ear deafness and chronic nephritis. So

far it has been clearly shown by historical, clinical and pathological proof that a hypercholesterinemia forms a basic link in the chain of diseases we have discussed. The only additional proof available would be experimental. For this reason it was attempted to produce a hypercholesterinemia in rabbits in order to determine if an artificial hypercholesterinemia could produce the same histopathological changes that we described in our nineteen histologically examined cases.

We[12] fed chemically pure cholesterin to twenty-six rabbits, whose average age at time of starting was three months. Feedings were given by the stomach tube. Ten animals at random were castrated, in order to determine how much the hypercholesterinemia is increased by this measure. The cholesterin was put in oil, 20 gm. of cholesterin

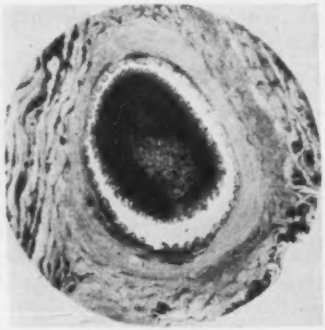

Fig. 9.

Fig. 10.

Fig. 9. Intima of internal carotid artery shows lipoid deposit in cholesterin-fed rabbit. This is quite similar to an arteriosclerosis of the vessel.
Fig. 10. Modiolus of cholesterin-fed rabbit showing vessels filled with lipoid.

to 100 c.c.m of oil. The animals were fed three times a week, starting with 5 c.c.m. and rapidly increasing it to 10 c.c.m per feeding. We fed sixteen of our animals for more than 100 days. The average feeding time was 70 days. The longest time was 227 days which animals received 184 gm. of cholesterin. Many animals died suddenly during the feedings. We killed sixteen animals immediately after feeding and allowed five of the 227-day animals to live for three months after the last feeding. The reason for this exception was to investigate to what extent a long-produced hypercholesterinemia is transient. Macroscopically, the arcus lipoides corneae gradually disappeared, and microscopically, the fatty deposits seem actually to have melted away.

The normal blood serum cholesterin of rabbits swings between .05 gm. and .09 gm. per 100 c.c.m. The repeated examinations of our rabbits revealed that in as short a time as fourteen days the average cholesterin content was 1.2 gm. per 100 c.c.m. and at 60 days it was 2.0 gm. The castrated rabbits showed a definite proportional increase.

We examined the blood pressure in five of our animals at random and were able to confirm Westphal's[4] finding that artificial hypercholesterinemia is accompanied by hypertension.

The temporal bones were fixed and examined with the identical technique used in the human specimens. The control animals showed no fatty or lipoid changes in the ear.

Our group of sixteen rabbits all showed practically the same histopathological changes with a difference of degree proportionate to the period of time of cholesterol feeding.

Cerumen in the external auditory canal showed a high degree of lipoid deposit. This may or may not be within normal limits. The cartilage of the external ear, however, showed a tremendously high degree of lipoid in droplet formation. The nuclei of the cells are almost obliterated.

The drum membranes show a quite characteristic fatty deposition with a diffuse lipoid deposit in all parts of the drum (see Fig. 6). The mucous membrane of the middle ear shows scattered lipoid deposits but the capillaries of the mucosa contain a serum heavily laden with lipoids.

The bone marrow of the ossicles and the ligamentous joints show, just as they do in the human, a slight deposit in the peripheral parts and a more marked diffuse one about the annular ligament of the stapes (see Fig. 7).

The secondary tympanic membrane shows no definite changes. The remainder of the bony parts show a marked lipoid increase in the marrow and the capillaries are filled with serum containing lipoids.

Muscles and connective tissue show a fine fatty droplet deposition.

There are no changes to be seen in the facial nerve itself but the bony cells of the facial canal show an annular deposit similar to that seen in the human sections. There are no deposits in or about the membranous and bony semicircular canals of the rabbits, which differs from the ring-like deposits noted in the human.

The cochlea shows marked fatty deposits in almost all parts (see Fig. 8). The deposit is most marked in the stria vascularis, the ligamentum spirale, the sulcus spiralis externus and in the cells of Claudius, Hense and Deiter. Reissner's membrane, the ductus cochlearis, the ganglion spirale and Rosenthal's canal show only sparse infiltration. The intima of the internal carotid artery shows a marked fatty infiltration (see Fig. 9). The modiolus of a cholesterin-fed rabbit shows the vessels filled with lipoid (see Fig. 10).

The animals that were killed three months after the last feeding show little or no histological evidence of lipoid deposit.

The experimental evidence obtained from an artificial hypercholesterinemia establishes clearly that cholesterin metabolism plays a large role in certain types of internal ear deafness.

In conclusion, it is the writer's belief that the systematic clinical, anatomical, pathological and experimental investigations presented in this paper establishes the importance of cholesterin metabolism and supplies a probable clue to the missing link which connects arteriosclerosis, essential hypertension, apoplexy and internal ear deafness.

Author's note: The original illustrations for this article were made in color (lumière) plates which show differentiation very much more clearly than the black and white reproductions seen in this article. Indulgence is asked if the reader is unable to make out the details as outlined in the text.

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THE DEVELOPMENT OF THE NOSE: ITS DYNAMIC RELATION TO TRAUMATIC INJURIES AND TO SUBMUCOUS RESECTION OF THE NASAL SEPTUM.*

Dr. William Wesley Carter, New York.

An intimate knowledge of anatomy is, of course, essential to the surgeon in whatever field he may operate. In addition to this, those of us who specialize in *rhinological surgery* must be equally familiar with the developmental forces that are responsible for the distinctive structure of the nose. This knowledge is necessary not only because the nose is an important respiratory organ and the seat of the sense of smell, but because of its esthetic value to the individual. The old adage, "An inch off the end of a man's nose makes a lot of difference," was born of a tragedy and did not have its origin in a beauty parlor. A fair proportion of the patients I operate upon for deformities of the nose are men, many of whom claim not to care a rap about esthetics, but who look upon disfigurement as a business handicap and for this reason seek relief.

The development of the nose involves dynamic problems that are intricate and the longer I study these problems the greater is my admiration for the wonderful provisions of nature that direct the interlocking, delicately poised forces that result in the construction of a symmetrical and properly proportioned nose. To a very large extent the process is a mechanical proposition, subject to the dynamic laws that govern the construction of a similar figure from any inanimate material.

Each side of the nose constitutes practically an independent dynamic entity. These separate units are dependent to a degree upon the uniform adjustment of two distinct mechanical units in order to develop a perfect nose. All nontraumatic deviations from the normal in the frame work of the nose record a maladjustment of these forces.

In order to understand the forces with which we have to deal, the adult nose should be considered a protruberance that has sprung from a more or less flat surface and that the process by which this was effected was, to a certain extent, analogous to that of erecting a tent.

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the septum corresponding to the tent pole and the soft tissues and nasal bones to the canvas. This rough analogy will serve to remind the clinician of the mechanical part played by the framework in the development of the organ. It must be kept clearly in mind, however, that since we are dealing with living tissues, the mechanical results may be modified by various constitutional conditions, by diseases, such as syphilis, atrophic rhinitis, etc., and by such intercurrent happenings as traumatism, the development of adenoids, etc.

From an histological point of view, the embryology of the nose does not interest us as clinicians, the physical phenomena, however, presented by the growing fetus, are not only interesting, but they

Fig. 1.

Fig. 2.

Fig. 1. Deformity—resulting from injury to nose in early infancy. Boy is now fifteen years old and defect in development was not noted until he was six years old.

Fig. 2. Girl thirteen years of age showing effect of atrophic rhinitis since early infancy on the development of the nose. This patient was Wassermann negative.

throw considerable light upon subsequent developments and the condition with which we, as operators, have to contend. You will recall that in the early fetus there is only one nasal cavity and this is not separate and distinct, but opens directly into the mouth. The division of this by the primitive vomerine cartilage begins before its separation from the mouth is effected by the palatal processes of the superior maxillae and the horizontal plates of the palate bones. A tripartite juncture of these planes in the median line is effected about the eighth week of fetal life. Fusion between the edges of these three segments, i. e., the two palatal processes and the vomer, begins forward and progresses backward and is complete about the tenth week.

Ideal development of the nasal cavities, the nasal septum and the palatal arch demands that there shall be perfect synchronism, both as to the time that the edges of these planes meet in the median line to form the nasal cavities and complete the palatal arch, and the rate of fusion and ossification at the point of meeting. It is unnecessary for me to emphasize the delicacy of the problem involved or to express surprise that anatomical symmetry is ever attained. So interesting have I found this problem that for many years I have noted in all of my patients the appearance of the palatal vault at this suture. I may say with due conservatism, that in 50 per cent of these there is anatomical evidence of lack of synchronism in either the time of fusion or the incidence and rate of ossification. This evidence consists chiefly in incomplete union (cleft palate), in inequality in the level of the two sides of the hard palate and more frequently and having still greater significance, is a bony ridge in the roof of the mouth. This is frequently nodular and extends anteroposteriorly along this suture. This is indubitable evidence that the vomer, which, in the process of raising the bridge of the nose, is under continuous vertical pressure, has pushed down between the palatal processes of the superior maxillae. I have, on several occasions, removed subperiosteally, this lower edge of the vomer where its protrusion into the roof of the mouth was causing the patient discomfort. I may state here that great care should be exercised in performing this operation, lest an opening be made into the nasal cavity above. Such an opening might be difficult to close.

The object of the thoughtful operator is to secure results that will be satisfactory, not for a week, a month or a year after the operation, but which will be permanent, both from a functional and esthetic point of view. Such results, especially in the case of adolescents, cannot be secured, unless the operator understands the laws of mechanics that are involved in this situation and has gauged in advance the direction, the energy and the duration of the forces employed by nature in the development of the flattened nose of the infant into the more prominent and shapely organ of the adult. Keeping this knowledge constantly before him, as a working guide, he must have sufficient manual dexterity to avoid interference with these forces. Precautions of this nature are probably more necessary in the field of rhinology than in any other part of the body, for the reason, as I have already stated, the development of the nose is largely a mechanical achievement and the laws of mechanics must be observed.

At birth the nose is flat and broad and is practically on a level with the cheeks. The nostrils are round and look forward. The

septum is short and relatively thicker than in the adult. The perichondrium is thick and inseparable from the cartilage, which is very thin and extremely flexible; the vomer, however, is well ossified. At birth the septum is always straight.

The anterior edge of the vomer inclines forward to the anterior nasal spine and is practically parallel to the prospective adult nasal bridge. This circumstance is of importance, for this bone, which is a vital factor in the development of the nasal bridge, has the great advantage of being able to act at right angles to the resistance that must be overcome through the medium of the septal cartilage and the vertical plate of the ethmoid. Then, too, the shape and position of this bone favors its strength as a foundation and support for the nose. The vomer not only constitutes the basic foundation for the constructive forces, but to a very considerable extent it is one of these forces.

This bone is composed of two compact layers of bone, fused below, but separated above to form a groove for the reception of the rostrum of the sphenoid behind and the septal cartilage in front. The lips of this groove, owing to imperfect ossification, may be widely separated and the interval may be filled with cartilage, or it may form a cavity between the two laminae: I have noted such a cavity in two of the cases of wolfnose I have operated upon. If the laminae of the vomer are not properly fused, so as to form a substantial basis upon which the cartilaginous septum may rest, a flat or saddleback deformity of the nose is certain to develop. If there is no fusion, the hideous deformity, known as wolfnose results. Here the nose is very broad, almost level with the face and the two sides of the organ are separated by a deep sulcus.

The ossification and growth of the vomer are not only interesting to follow, but much light is thrown upon the process of nasal development.

About the second month of fetal life a nucleus appears on either side in the back and lower part of the membrane which covers the vomerine cartilage. From these centers of ossification are developed the primitive lamellae which gradually become fused together to form one layer of bone. The fusion takes place behind and below, about the third month, and, as it extends forward and upward the cartilage between them is absorbed and a groove is formed in which the septal cartilage is lodged. Complete fusion between these lamellae of bone is not effected until puberty. It is easy to understand, therefore, how essential it is that the vomer should develop normally and that it should be preserved in a healthy condition during the years of adoles-

cence, if the nose is to be symmetrical and adapted to its facial environment.

The fact that the two plates of the vomer do not become fused into one until puberty and that this period, as a rule, coincides with the full development of the nose, is positive evidence of its importance as a constructive factor. Conversely, imperfect development of the vomer, impairment of its vitality, or its destruction in early life is always followed by saddleback deformity. The atrophic rhinitis, so frequent in these cases is, as I pointed out many years ago, the cause of the deformity and not the result, as is thought by many. Atrophic rhinitis is essentially a bone disease, for its chief pathology is manifested in the deeper layers of the periosteum. In this disease the normal development of the vomer is interfered with and therefore this bone does not properly perform its function of raising the bridge of the nose and the latter remains at its infantile level.

The vertical pressure exerted by the growing vomer is transmitted to the base of the skull and the bridge of the nose through the vertical plate of the ethmoid and the cartilaginous septum. This pressure is continuous and it increases in intensity from the second year up to puberty. I have noted that in some patients the vertical tension is great even up to the thirtieth year. This fact is of clinical importance not only in operations upon the septum, but in injuries to the nose where the nasal bones have been fractured or dislocated, for the cartilaginous septum which may not have been injured, owing to its elasticity, may push the movable fragments of bone aside and appear as a ridgelike hump on the dorsum of the nose. In many of these cases it is necessary to shave off the upper edge of the septum in order to reduce the fracture and secure a straight nasal bridge: I do this from within the nose, so there is no external scar.

Long ago I first called attention to the inherent vertical tension of the septum during adolescence as a cardinal factor in the production of deviation of the septum as well as twists and other asymmetries of the external nose.

Deviations of the septum do not occur prior to the second year, but from this age on to full maturity they are seen with increasing frequency. In the Caucasian race about 75 per cent of the adults are affected. On the other hand, in the Mongolian and Negro races, where the flat, broad, infantile nose is an easily recognized characteristic, the so-called idiopathic deviation is seldom, if ever, seen. This distinction, itself, calls attention to the importance of the septum as a factor in the elevation of the nasal bridge, for the nose in these races has retained its infantile type and there is no evidence of the

septum having been under the vertical strain necessary to produce the nasal prominence characteristic of the white races.

In summarizing my observations, which have been drawn from practical clinical experiences and from following up my cases over a period of many years, I will say:

1. That the development of the nose is largely a mechanical proposition.

2. The septum is the most important factor in the elevation and subsequently in the support of the nasal bridge; consequently every operator should be ever mindful of the fact that neither cartilage nor bone, removed from the septum, is ever replaced, and that conservatism should always be practiced in doing the submucous operation.

3. In the treatment of traumatic injuries, where it is possible, replacement of the fractured and dislocated parts should be practiced. This is imperative in the young subject, otherwise a deformity is certain to develop, though it may not become noticeable for several years.

In many of these cases I have found my bridge-splint useful; it is not applicable, however, in the treatment of very young children.

4. In developmental deformities, an effort should be made to adjust the parts so that the lines of force shall be so directed that they will construct a normal organ.

5. If there is a deficiency in material for reconstruction, autogenous bone and cartilage transplants from the rib should be used in the manner first advocated by me. Under no circumstances should foreign materials, such as paraffin, celluloid, ivory, etc., be used for this purpose, as they are not safe nor do they remain permanently in the living tissues.

2 East 54th Street.

THE CONTROL OF POSTOPERATIVE EDEMA OF THE NOSE AND THROAT.*

Dr. Leon Felderman, Philadelphia.

This article considers postoperative edema of the nose and throat from the standpoint of the two established surgical procedures in those areas. I have selected the classical submucous resection and tonsillectomy.

It would be a tiresome task for my auditors if I were to attempt to submit before you the results of my observation of the great number of operations which fall within the domain of the otolaryngologist. However, I will stress edema of the nose and throat. Since edema can be checked in these two classes of operations, it is logical to believe that the same treatment can be applied to other operations of the upper air passages. If I speak in interchangeable terms of the aforementioned operations, I hope you will understand.

We are acquainted with the facies of the patient who had submitted to a deflected septum operation. Apart from the bleeding and occluded nostrils which is a very uncomfortable situation for the patient, the condition of the nasal blocking continues for many days after the operation, due chiefly to edema and superimposed false membrane. The patient having in mind that so many bones had been resected is somewhat bewildered that his nasal passages do not remain patent immediately and will often look for an explanation of his surgeon. On the other hand, when a patient submits to a tonsillectomy the picture of distress is more striking. Here we have a subject who was relatively normal the day of the operation, but once he had submitted to the mysteries of tonsil surgery, he feels that true information was withheld. To paraphrase one of the common advertisements, "The pain and discomfort is long remembered after the fee is paid," which in some cases is quite a long time.

When a patient has his tonsils removed the mechanical forces of deglutition is seriously compromised, so much so, that even the imbibition of liquids is a struggling, painful procedure[1]. The physical factors of speech mechanism are impaired. The intensity, pitch and resononce are thrown out of alignment so the orderly process of speech is no longer physiologic but pathologic. The swollen uvula

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adds materially to this distressing feature and rhinolalia aperta helps the discomfort along. What happens is that the tissues of the throat have become "water-logged" so that the act of communicating with the outside world by means of vocal mechanism is temporarily impaired.

Claude Bernard, the great physiologist, in 1878 published his first monograph on this interesting subject. In the thesis "lecons sur les Phenemones de la vie," he gave the medical profession practically all that we know today of edema. Research workers like Krogh, Holdane and others have enlarged his viewpoint without changing the basic facts expounded by Bernard. Internists point to edema as a condition following certain nutritional disorders. This condition was widespread during the world war. The writer has been in communication with subjects on the Amiens sector who showed unmistakable effects of semistarvation diets over a long period of time and were referred to as war dropsy. M. H. Barker, of Northwestern University Medical School, has used control experiment with animals and was able to simulate this condition by reducing the protein element.

The modern concept of edema is viewed as a disturbance in the distribution volume of the body fluids. Endocrinologists formerly believed that an immediate alteration takes place through the thyroid and pituary glands.

An interesting paradox takes place when a patient has his tonsils removed. First a gradual general anhydremia appears; that is to say, a depletion of the body fluids; but *in situ* where the surgeon's scalpel was wielded the entire oropharyngeal cavity appears to be overrun with fluids and together with a swollen uvula the orderly march of events which controls the act of swallowing and speaking is strongly interfered with. This mechanical stasis, although a temporary condition, can be eliminated entirely.

The local shift-producing edema is due to disturbances of chemicophysical laws affecting the exchange of body fluids between tissue spaces and capillaries. This results in serving as a buffer to the injured parts. Starling successfully demonstrated the shift of fluids which he called osmotic equilibrium. Other writers prefer to name it as osmotic dis-equilibrium. The influential role of the lymphatic system must not be discounted, since the function of the lymphatics does not only favor absorption and regulates the lymphatic flow but acts as a sieve by separating the protein matter from the adjacent tissue fluids. The occurrence of edema in certain types may be regarded as beneficial inasmuch as it helps to regulate the volume of blood.

The problem of edema has been attacked from many viewpoints. Numerous theories have been advanced to explain its mechanics. I have endeavored to steer clear of the various forms of edema and limit myself to the postsurgical type, such as falls within the province of the otolaryngologist.

Before I enter into the control phase, I may do well to postulate a few mechanical factors which govern this pathological condition. Under normal circumstances the human blood is maintained at certain constant volume and pressure.

When a patient is subjected to an operation there is a sharp decline of the plasma proteins due to the primary hemorrhage and the nervous reaction also figures into the composite picture of edema.

The mechanistic factors can be mentioned as follows: *1.* Nervous reflexes are increased. *2.* Increased capillary permeability. *3.* Increase in hydrogen ion concentration and increased affinity of tissue colloids for water. *4.* There is a tendency to acid base equilibrium.

Dr. J. B. Collip in his controlled animal experiments has been able to demonstrate the importance of a potent hormone as calcium metabolism. A short time later Greenwald and Gross cleared all doubts on the subject by removal of the parathyroid glands in dogs, first studying their normal behavior and then supplying the body with an extract of parathyroid hormone, restoring these animals to their normal condition. It was then shown that calcium salts diminish the permeability of the blood vessels and its therapeutic employment in inflammatory types of edema was endorsed.

J. H. Hamberger demonstrated that there is a strong interaction between calcium, sodium and potassium ions in a perfusing fluid by bringing about a constriction of the walls of the capillaries and reducing their permeability. "According to Blum, calcium administration diminishes the sodium of the blood and prevents it from migrating from the blood into the inflammatory area. As the current of water follows sodium, the accumulation of fluid which ordinarily accompanies the inflammatory process is inhibited."

Calcium salt seems to favor the coagulation of blood by increasing the thrombin ferments in the body fluid. The exact mode of operation on the blood, to some observers, is still a disputed question.

Prior to operations on the nose and throat I always make it the invariable rule to administer to the patient by mouth a few tablets of calcium gluconate (Sandoz); children receiving a proportionate dose, which they take without difficulty because of its pleasant taste and resemblance to chocolate. Thirty minutes before the operation I order 1 c.c. ampoule parathormone (Lilly), which is equivalent to

20 units and it is administered intramuscularly. The results are most astonishing.

In nose operations, there is almost a total absence of secondary membrane formation. The bleeding is lessened and the patient's nostrils remain clear during the healing period. In cases where tonsils are removed the edema condition of the throat is absent and the patient can carry on the acts of deglutition and speaking with a great deal of freedom and comfort. The patient is free from reflected pains in the ears which sometimes follow tonsil operation. When the tongue is coated and swollen, not infrequently I give the patient a tablet composed of caroid powder, to be dissolved on the tongue and repeated every two or thre hours. Hypercalcemia has not been experienced by any of my patients, since this form of calcium medication is given only a short time.

Gastrointestinal disturbances would manifest themselves if a patient should take large amounts of calcium. Once the line of tolerance is passed the drug can always be withdrawn; however, one must bear in mind the exaggerated responses from the ingestion of calcium salts. For instance if the parathyroid hormone is given too much and the calcium in the blood is increased above the normal level other symptoms may intervene, such as atonia, profound depression, atopia, vomiting, bloody stools and, although rare, death may ensue. In doubtful cases, A. T. Cameron[3] suggests the chemistry of the blood should be studied; for there is no antidote for it if the patient develops hypercalcemia. A. W. Hueper[4], of Loyola University School of Medicine, has shown that metastatic calcifications of other organs of the body can take place, such as thyroid, lungs, heart muscle, stomach, duodenum and kidneys. In view of his experimental results the otolaryngologist can benefit by careful employment of parathyroid hormone, according to Hueper an increase of blood calcium, 10-15 m.g. per 100 c.c. of blood, can be tolerated by the patient without exhibiting any of the above aforementioned symptoms.

SUMMARY.

1. Edema is caused by increased capillary permeability as will increase affinity of the tissue colloids for H_2O.

2. Primary hemorrhage in nose and throat operations favors the mechanical escape of fluid into the surrounding structures where the operation took place.

3. The administration of calcium salts and their derivatives in judicious doses diminishes the permeability of blood vessels, decreases the surgical edema.

4. The administration of calcium salts.
5. Checks postoperative hemorrhages and prevents acidosis.
6. Hastens postoperative convalescence.

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RETROBULBAR ABSCESS COMPLICATING SUBMUCOUS RESECTION.

DR. PAUL L. MAHONEY, Little Rock, Ark.

A retrobulbar abscess following a submucous resection seems to be a complication that is rarely encountered. A reference to this condition was not found in a recent search of all available literature. I hope that I will not have the opportunity to report a second case.

Mrs. S., age 23 years, well developed, with a general appearance of good health, first came to our office Nov. 2, 1928. Her complaint was an inconstant headache, dull in character, of several years' duration, having its origin in the left frontal region and radiating to the region of the left occiput.

On examination the ears were found to be negative, the perpendicular plate of the ethmoidal bone deviated to the left, making inspection of this region impossible. The mucous membrane was normal in appearance and there was no evidence of infection noted. The pharynx and nasopharynx was negative. The tonsils were absent and the teeth were in excellent condition. Transillumination and X-ray of the sinuses was negative. Examination of the eyes was made and they were normal in every respect. Cocainization of the

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sphenopalatine ganglion gave no relief. General physical examinations were negative. A submucous resection was recommended and refused.

Jan. 8, 1929, she returned to our office and examination on this date revealed the usual findings of acute coryza. This readily subsided and subsequent examination failed to reveal any pathology.

Sept. 11, she returned for operation and a submucous resection was done the following day. The left middle turbinate was infracted and inspection of the ethmoidal region failed to reveal any evidence of infection.

Sept. 14, she complained of severe headache and difficulty in breathing. Examination revealed a complete nasal blocking. The next day there was noted slight edema with inflammation of the left lids. The temperature was 99.6° F. This condition progressed to the extent that on Sept. 17, there was present marked edema, inflammation, protrusion of the eye ball, with a temperature elevation of 103° F. The chemosis and protrusion became so extreme that for several days it seemed that the loss of this eye was eminent. It was impossible to reduce the nasal blocking and narcotics were necessary to control the headache and pain at this time.

She was admitted to the hospital this date for operation. An incision was made at the inner angle of the left eye, the periosteum was elevated to a point near the entrance of the optic nerve where several drops of pus were found. There was no evidence of any communication with the nose. A rubber tissue drain was inserted and the wound allowed to remain open. At all times the cornea was carefully protected. For several days the temperature ranged from 99° F. in the mornings to 102.3° F. in the afternoons. The highest white cell count was 10,000 and several blood cultures were negative. The urine remained negative throughout.

Oct. 1, she was discharged from the hospital with a normal temperature, the protrusion was diminishing, and the drainage but slight. During her convalescence diplopia was noted. She made a complete recovery, the diplopia ceased and normal vision was regained. At this time she continues to be free of headaches, and nothing remains as a reminder, but a small unnoticeable scar.

831 Donaghey Bldg.

NEAR-FATAL POSTOPERATIVE SECONDARY NASAL HEMORRHAGE.*

DR. LOUIS R. EFFLER, Toledo, Ohio.

It had not been exactly our outward boast but it had been certainly our inward feeling that any hemorrhage of nose or throat character must surrender quickly to our skill. The following two cases, however, have reduced us to a more humble viewpoint. Entirely unlike anything previously experienced in a 15-year practice, they exhibited the peculiar coincidence of appearing almost in successive weeks and in successive cases.

CASE REPORTS.

Case 1: Margaret B., aged 27 years. July 10, 1931: Patient first seen with cold in head and pain in right frontal region. Temperature 99.5°F. History of frequent head colds and complete obstruction to right nostril by a badly deviated septum. Observed and treated off and on for about six weeks.

Aug. 29: Submucous resection performed with left middle and inferior turbinotomies. Operation uneventful. Packing removed 36 hours later. Moderate bleeding, left; checked spontaneously within 10 minutes. First hemorrhage on the fifth day postoperative; moderate; checked spontaneously. On twelfth day postoperative, second severe hemorrhage. Checked with nasal packing by another doctor; hospitalized. Seen by me several hours later. No bleeding; packing removed, no bleeding. Discharged from hospital four days later.

Sept. 18, or the twenty-first day postoperative: Third severe hemorrhage. Packed and partly checked by another doctor; hospitalized.

Sept. 19: Fresh hemorrhages at 7:30 A. M., 9:30 A. M., and 7:00 P. M., despite anterior packing and a postnasal plug. Repacked and replugged; dry. Intravenously 200 c.c. Ringer's solution with 5 per cent dextrose and Murphy drip.

Sept. 20: Uneventful; packing intact; only moderate ooze.

Sept. 21: Packing and plug removed; renewed violent bleeding; repacked and replugged; bleeding checked; transfusion, 500 c.c. whole blood.

Sept. 22, 8:30 P. M.: Postnasal plug removed, no bleeding; right nasal packing removed, no bleeding; left nasal packing partly removed, brisk bleeding encountered; remainder of packing left in situ, dripping soon stopped. 12:30 P. M.: Moderate seepage of fresh

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blood into throat; checked spontaneously. 4 P. M.: Major hemorrhage from both nostrils, despite packing in left. Repacked and replugged; checked. 6:30 P. M.: Second transfusion, 500 c.c. whole blood.

Sept. 23: Packing not disturbed; no bleeding; note daily trend of temperature upward. Circa 103°F.; discount post-transfusion rise to 105.5°F. Patient very toxic. At 4:30 P. M., postnasal plug removed; no bleeding.

Sept. 24, 8:30 A. M.: Anterior packing carefully removed; moderate ooze but no frank bleeding from this point.

Oct. 11: Patient discharged from hospital.

Case 2: Marie D., age 28 years. First seen by me in 1929, with an acute suppurative left frontal sinusitis. Relieved by left middle turbinotomy and left frontal lavage.

Sept. 16, 1931: Recurrence of severe left frontal pain with temperature 99.5°F.

Sept. 19: At patient's own request performed open operation on left frontal and left anterior ethmoid sinuses (Moure's operation), local; much frank pus and polypoid tissue encountered. Wound sutured; no packing; no bleeding.

Sept 25: Uneventful convalescence to this point. Sudden severe nasal hemorrhage. Packed and plugged by local doctor; hospitalized. Despite packing, four more severe hemorrhages before seen by me. Repacked and replugged by me; bleeding checked.

Sept. 26: No bleeding. Eyes swollen shut, with marked swelling of forehead. Healed incision reopened for drainage; released profuse bloody pus.

Sept 27: Packing and plug removed; profuse bleeding; repacked and replugged; bleeding checked.

Sept. 28: Profuse bleeding despite packing and plug. Repacked and replugged; checked. First transfusion of 400 c.c. citrated blood.

Sept. 29: Postnasal plug removed; no bleeding.

Sept. 31, 9 A. M.: Right anterior nasal packing removed; no bleeding. Temperature 105.5°F. 4:30 P. M.: Left anterior nasal packing removed; no bleeding. Temperature 102.5°F. Looks septic; face edematous around eyes, nose, and forehead.

Oct. 1, 12:10 P. M.: Minor hemorrhage from left naris and large fresh clot removed from postnasal space. 7 P. M.: Major hemorrhage, both nares and into throat. Simpson intranasal tampons inserted, left naris; bleeding checked in several minutes. 11 P. M.: Major hemorrhage. Removed old Simpson plugs and reinserted fresh ones. Again checked.

Oct. 2: Three more minor hemorrhages in course of night, through Simpson plugs; checked spontaneously and no interference. 8 A. M.: Major hemorrhage through Simpson plugs; removed; fresh plugs inserted. Bleeding checked within 10 minutes. 3:30 P. M.: Fresh bleeding, 8-10 drops per minute. Stopped spontaneously in 10 minutes. Large fresh clot removed from pharynx. 4:30 P. M.: Major hemorrhage through Simpson plugs. Discarded in disgust. Replaced with gauze packing; bleeding not checked until pressure was used with finger and thumb across bridge of nose for 10 minutes. 6:30 P. M.: Second transfusion of 200 c.c. whole blood.

Oct. 3: No bleeding during day.

Oct. 4: Packing removed from nose; no bleeding; no further bleeding from this point.

Oct. 11: Patient discharged from hospital.

COMMENT.

1. *Primary Infection:* One of the first outstanding points in common in these two bleeding cases is that they were both primarily infected. Each showed a low grade temperature. The average head cold is seldom capable of producing temperature. An influenzal infection, however, frequently shows this mild fever reaction.

Proper respect was shown this infection in Case 1. Operation was delayed fully six weeks on its account. This was deemed sufficient time for the acuteness to subside. That it failed of its purpose is beside the mark. Proper consideration was given the infection also in Case 2. Unfortunately, the severity of pain permitted only a few days' delay before operation. Whether the result would have been different had there been a longer wait, it is impossible to say. Hindsight seems always better than foresight.

We cannot help but believe that the infection was the causative factor of bleeding in both cases. It is easy to visualize in the healing process the raw surfaces bathed by irritating discharges until a slough leaves a large vessel exposed. The operative technique itself seemed faultless, otherwise there would have been primary bleeding at operation.

We distinguish *primary* infection here from *secondary* infection. The secondary infection feature will crop up later as an important factor in the near-fatalities.

2. *Secondary Hemorrhage:* It is almost a rule that secondary hemorrhage is seldom severe. At least it is more easily controllable than primary hemorrhage. The longer it is delayed the easier it is to control.

Here, however, was no simple bleeding. Here were two cases with the severest bleeding, either primary or secondary, ever seen in our experience. In each case there was danger of exsanguination. The bleeding did not consist of a simple ooze or a slow drip. At certain stages, blood poured out of both nostrils as from open taps, while at the same time mouthful after mouthful was spit out, to add to the excitement. It is disconcerting to attend to three "leaks" at the same time. It is quite a bit more disconcerting, however, to "solder" up three leaks one after the other and then have them all continue to leak almost as briskly as before.

This means of course only one thing. It means *faultiness of packing*. Of course, the packing was faulty for a number of good reasons: *a*. The copiousness of the blood flow. *b*. The blindness of the field. *c*. The dread and discomfort on the part of the patient from repeated packing. *d*. The fear and apprehension on the part of the patient from repeated bleedings. *e*. The speed required to check the appalling loss of blood.

Undoubtedly, in each case a large artery was involved. Peculiarly, in each case it was in the left naris. The exact location, however, of this bleeding artery was for a long time impossible to discover. The field was always completely obscured by blood or packing. When at last the probable location in each instance was discovered, sensible progress was made at once toward permanent stoppage.

In Case 1 the vessel was located at the posterior end of the left inferior turbinate (probably the posterior lateral branches from the sphenopalatine). In Case 2 the vessel was located at the external wall of the nasofrontal trough, *probably the left anterior ethmoidal*. In any case, pressure packing directed at these two points produced eventually collapse of the vessel walls long enough to permit organization and healing.

3. Packing: Regardless of early failures in these cases, the rule still stands that proper packing will stop *any* nasal hemorrhage. The difficulty is to secure proper packing in the face of the difficulties above enumerated.

Various *methods* of packing were employed in these cases. At first, simple anterior nasal packing was sufficient on the bleeding side. When this appeared inadequate, anteroposterior packing was effected on the bleeding side with long-bladed Killian forceps. Later still, anteroposterior packing of both sides was found necessary to secure the effect of counter-pressure from the nonbleeding side. Finally, the anteroposterior packing of both sides with the insertion of a postnasal plug was found essential.

Various *types* of packing were employed. First dry one-inch gauze was used. Then dry gauze saturated variously with fibrogen, cocain and adrenalin, etc. Needless to say, the styptics and coagulants were found almost useless. Their use is reserved for capillary bleeding. Later vaselined gauze was used. At one point Simpson's intranasal tampons were tried. Whatever their value in minor hemorrhage they will prove disappointing in major hemorrhage cases. The bleeding was checked for several hours by them in Case 2. In each instance, however, profuse bleeding took place after a quiet period, probably *around* them, to indicate their inadequacy. In the interval necessary for their swelling, a clot probably formed between them and the lateral nasal wall. This clot seemingly interfered with complete collapse of the bleeding vessel. It still permitted enough oozing to take place, which then burst forth into fresh bleeding as soon as sufficient pressure was built up. In other words, the temporary check was more apparent than real. The greatest reliance may be put in these cases in simple gauze-packing properly placed.

4. Secondary Infection: In addition to the hazards of repeated profuse hemorrhage and the probable danger of exsanguination, there is added the ever-present worry from secondary infection. Every rhinologist knows that it is dangerous to leave a nose packed for more than 36 to 48 hours. The dammed-up secretions, especially if infected, will invade new areas. These areas are usually the sinuses or the Eustachian tubes with resulting otitis media. Far more serious, however, is the resorption of these toxic products into the general system. More serious still is the invasion of the lymph vessels and the blood stream by the bacteria themselves. Added to the secondary anemia from severe loss in blood volume, there is the added dread of sepsis.

Let us see whether these dangers are over-emphasized in these cases. It must be assumed that the nasal packing could not be removed permanently without a renewal of hemorrhage even though the presence of the packing predisposed to toxemia or worse. The cases resolved themselves, therefore, into a race with death. It became a simple question of whether our patients might die before the packing could be removed or whether the packing could be removed in time to permit them to live.

Case 1 showed no *local* effects from prolonged packing. The anteroposterior packing was continued for six days and the postnasal plug for five days. A *general* reaction, however, was noted in a steadily mounting temperature. At a given time on each succeeding day, the rise was approximately half-a-degree to a high point of

103°F. At this point the packing was permanently removed and the temperature began to subside by lysis. The hemoglobin went to a low of 44 and the R. B. C. to a low of 1,800,000 per c.c. The W. B. C. went to a high of 17,000 per c.c. Despite her transfusions, this patient had tapped her reserve through loss of blood and toxic resorption. Her condition was grave in the extreme.

Case 2 showed two *local* effects from prolonged nasal and postnasal packing. The first was a severe edema that swelled shut both eyes and extended far up the forehead even in the first 48 hours. A cavernous sinusitis was at once suspected in view of the known suppurative left frontal sinusitis. The healed wound over the left frontal was slit open as the only means of drainage.

The second local effect of prolonged nasal and postnasal packing was a red, bulging left ear-drum on the seventh day. This was paracentesed.

The *general* effects, however, were even more awesome. Coincident with the swelling shut of both eyes, there occurred a chill and two temperature rises to 105°F. and 105.5°F., respectively, on successive days. These findings lent some credence to the fear of cavernous sinusitis or possibly meningitis. In addition, the blood culture was reported *positive* for staphylococcus albus. Despite the danger of such serious complications, the bleeding still continued and demanded the necessity for still further nasal and postnasal packing.

The anteroposterior packing was continued for nine days and the postnasal plug for four days. The Hemoglobin hit a low of 54 and the R. B. C. a low of 2,480,000 per c.c. The W. B. C. hit a high of 18,000 per c.c. No white count was taken during the extreme temperature rises. Two transfusions were also given this patient. Her reserve badly tapped, they helped her materially to ride on an even keel.

SUMMARY.

1. Primary infection of the nose or throat must be respected. Wherever possible, operation should be delayed until acuteness has subsided.

2. Secondary, and especially late secondary hemorrhage, is seldom severe. The rule, however, may be broken.

3. Proper packing will stop any nasal hemorrhage. There are difficulties in the way, however, of securing proper packing.

4. There is great worry from secondary infection in cases demanding prolonged nasal packing. To secondary anemia may be added the danger of sepsis.

222 Michigan Street.

AN EFFECTIVE METHOD OF CONTROLLING SECONDARY HEMORRHAGE.*

DR. J. JEROME LITTELL, Indianapolis.

The problem of secondary hemorrhage following nose and throat surgery is one that has received considerable attention in the literature, the most recent contribution being that of Early in a late issue of THE LARYNGOSCOPE. It is widely recognized as one of the greatest nuisances and worries that accompany the care of our convalescing patients.

My excuse in making this the subject of a paper is that I should like to recall to you an old remedy in a new and, to me, much more useful form. It is not my wish to claim originality as I am aware that "there is nothing new under the sun." Perhaps some of you have already used it. I am sure you have in its older form, but I have been unable to find the newer method mentioned elsewhere.

The cause of secondary hemorrhage has been variously described as: *1.* postoperative slough laying bare blood vessels; *2.* metabolic disturbances, *viz.:* menstruation, circulatory disorders, debilitated conditions; *3.* over-exertion or indulgences on the part of the patient.

In my experience as assistant and "trouble shooter" for a busy otolaryngologist, and later in my own practice, I have had at least fifty such cases following nose and throat surgery. Among these I include hemorrhage occurring between the first and fourteenth days following operation. The greater per cent we found between the third and tenth days. They began without warning and usually not following any unusual exertion. Quite a few of them began during quiet sleep, the patient being awakened by the presence of blood in the throat. Often the patient was in a debilitated state (and this seemed to be especially true of the nose cases). In only two cases did the erosion occur into a large vessel. It was quite reasonable to assume separation of a slough in many of them, but the repeated occurrence in individuals who were badly run down—such as the arrested tuberculous (three cases), has forced the conclusion that there is a systemic defect; furthermore, the blood vessels when once opened show an astounding tendency to continue bleeding. It is almost axiomatic that recurrence of the bleeding from the same or another point is to be expected, when any other than the one method

*Read before the Indiana Academy of Otology, Rhinology and Otolaryngology, Dec. 9, 1921, at Terre Haute, Ind.

of hemostasis which I am about to describe is used, excepting prolonged packing, which is quite undesirable and necessary only where profuse arterial bleeding is encountered.

As a case in point, may I quote the following: In June of this year I did a submucous resection and amputation of the anterior two-thirds of middle turbinate body on a young mother. As she had been through an unusually harrowing year from domestic complications she was glad to remain in the hospital five days for the rest. These, like the operation, were quite uneventful. On the seventh day she was brought in from her home, sixty miles away, with the story that while she was quietly resting in bed her nose had suddenly begun to bleed, with some vigor, from the right side (the side of the turbinectomy) and that a local specialist had found it necessary to pack both sides tightly to stop it. At the end of twelve hours I removed the pack from the left side for her comfort, with no untoward result. After twelve hours more I removed the pack from the bleeding point on the right. There was no trouble for about four hours, when the right side began to bleed from the anterior attachment of the middle turbinate body. Being temporarily out of the hemostatic upon which I rely, I was forced to pack again. (Attempt at cautery with trichloracetic acid was unsuccessful.) A few hours later the left side began to bleed from a raw area on the septum half-way back and had to be repacked. The following day my hemostatic applicators arrived and I approached the job of packing removal with some confidence. These consist simply of long wooden applicators tipped with fused beads of silver nitrate, 75 per cent, furnished by Tappanzee Surgical Co., Nyack, N. Y. I removed the packing cautiously from the left side. The area on the septum began to pour blood. I introduced an adrenalin soaked cotton pack, pressed it in place till the tissue blanched and the bleeding checked, then cauterized the area with the lunar caustic. The right side bled in like manner and was similarly treated. We had no more trouble, not even blood-tinged mucus.

SUMMARY.

This rather involved history is characteristic of these cases and brings out well the points I wish to make:

1. Delayed occurrence.

2. Debilitated condition of patient.

3. Tendency to recurrence following use of ordinary hemostatic methods.

4. Prompt efficacy of adrenalin plus silver nitrate stick.

This method of first checking the blood flow with an adrenalin soaked cotton sponge and cautery with silver nitrate stick is equally effective in post-tonsillectomy bleeding if the clots are completely removed and thorough applications are made. I have not yet seen it fail. As it has saved me many sleepless and perspiring hours, I always carry a supply in my grip.

I have not used surgical diathermy or a dessicating spark to cauterize these areas. I should imagine it might work equally well if the area were first made temporarily bloodless with adrenalin and care was taken not to penetrate too deeply so that a slough would follow its use. However, it lacks the convenience of caustic applicators and could hardly be more effective. I heartily recommend them to those who are unable at all times to keep their surgery bloodless.

These applicators are manufactured by the Tappan Zee Surg. Co., Nyack, N. Y., whose advertisement appears on page 5 of this issue.

603 Hume-Mansur Building.

A CASE OF CARCINOMA OF THE TONGUE (MISREFERENCE OF THE PAIN FOLLOWING ALCOHOL ANESTHESIA). REPORT OF A CASE.

Dr. David Mezz, Brooklyn.

The phenomenon of cortical misreference of pain, by which is meant a pain stimulus originating in one branch of a nerve being falsely interpreted centrally as coming from another and innocent branch of the same nerve, is a matter of daily occurrence. This, for example, is the mechanism behind the very frequent symptom of supraorbital pain accompanying antritis wherein the infraorbital nerve (or some other branch of the maxillary division of the trigeminal nerve) contains the truly stimulated nerve fibres but the pain is erroneously experienced by the supraorbital branch of the ophthalmic division of the trigeminal nerve by cortical misreference.

Our patient presented a different angle to the same mechanism, viz., cortical transfer of the pain from the mandibular division of

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the trigeminal nerve following alcoholic anesthetization of that division to the maxillary division of the trigeminal nerve:

Case, J. K., Male, 45, came to the outpatient department of Greenpoint Hospital, service of Dr. J. E. Braunstein, on April 10, 1931, complaining of: *1.* "Sore on tongue;" *2.* Severe continuous neuralgic pains in the left mandible, left ear, and diffusely throughout the left skull, aggravated by mastication and swallowing; *3.* Loss of strength because of the lack of food and sleep, both the result of pain. These symptoms had begun three months prior to the visit and were becoming progressively worse.

Upon examination, we noted a fairly well-nourished adult male with a pale, wan, fatigued face. On the left lateral border of the tongue, midway between the base and the tip, there was an ulcerated area, one centimeter in diameter, moderately deep, irregular and surrounded by a stony-hard indurated edge. The floor was irregular and covered by a foul, dirty, sloughing debris. Any movement of the tongue produced agonizing pain referred mainly to the left ear and left side of the head. No glands were palpable in the neck. The diagnosis of squamous cell carcinoma was substantiated subsequently by a biopsy report on a specimen taken at this first visit.

It was felt that nothing was more essential for immediate treatment than to eliminate promptly the pain which prevented both eating and sleeping and which was hastening a cachectic state.

The sensory nerves of the tongue are: *1.* the lingual nerve, a branch of the 3rd division (mandibular division) of the 5th cranial nerve, which is reinforced by the chorda tympani branch of the facial nerve to supply the anterior two-thirds of the tongue; *2.* lingual branches from the 9th cranial nerve supplying the root of the tongue including the vallate and foliate papillae; *3.* and the superior laryngeal branch of the vagus nerve supplying a small area near the epiglottis.

Our patient had his lesion well confined to the anterior two-thirds of the tongue, that portion innervated by the lingual nerve-chorda tympani combination. However, the chorda tympani component is essentially for the afferent impulses of taste; which therefore means that the mandibular division of the 5th cranial nerve by way of its lingual branch is the essential sensory nerve for pain coming from the anterior two-thirds of the tongue.

It was decided to anesthetize the mandibular nerve at its entrance into the mandibular foramen, using the same technic the dentists employ to gain anesthesia for the extraction of a lower molar tooth. One per cent novocaine was injected first as a test for the efficacy of the entire idea. Within fifteen minutes, the patient was completely

relieved of his pain for the first time in three months. We then injected 2 c.c. of 70 per cent alcohol and gained complete relief of the pain. The patient was now referred to the x-ray and radium department for treatment of the lesion.

Upon a subsequent visit two weeks later, the patient complained of a dull, neuralgic ache in the left superior maxilla and left upper teeth. This was in annoying contrast to the "dead feeling" in the left inferior maxilla and left half of the tongue. There was no objective reason in the upper jaw, nose, or teeth to explain this superior maxillary pain and thus we felt this to be a case of mis-referred pain.

Having obliterated the cortical impression of the pain stimulus in the mandibular nerve by our injection, the stimuli were still actively sufficient to furnish a central impression whereupon the center misinterpreted the peripheral origin of the pain as coming from the maxillary division; hence the pain in the superior maxilla and left upper teeth.

The dull neuralgic ache in the upper jaw was alleviated temporarily by cocainization of the spheno-palatine ganglion; it persisted for several weeks and then fortunately disappeared. The patient's relief from pain lasted one month during which he gained considerably in weight and strength. When last seen the pain was slowly returning. He has been referred to the X-ray and radium department.

125 Eastern Parkway.

SERIOUS THROAT INFECTIONS.

Dr. Harold Hays, New York.

It has been my misfortune to have had a great many patients during the past three years who have died from infections of the throat, of one type or another. In fact I have had more deaths from throat infections than from ear infections and their complications, although I have attended to many more ear cases. Such a statement may seem most surprising. However, in view of the fact that people pay far less attention to infections of the throat until they become quite serious, one can see the great possibility of such a condition getting beyond one's reach. Some of these conditions may respond to operative interference but a great many of them do not.

When one realizes the enormous area of mucous membrane in the oral cavity, pharynx, nasopharynx, hypopharynx and larynx, it is indeed surprising that serious infections do not take place more often. Over 25 years ago I proved in the chemical laboratory that salivary secretion in itself is slightly antiseptic and most certainly the mucus secreted by this vast area must also assist in washing pyogenic organisms away. However, a lowered resistance of the patient, either locally or generally, may tend to allow of serious infections taking place which can not be overthrown by the usual means at our command.

In general, we think of the tonsils as giving rise to definite infections throughout the system and we are therefore on the lookout for any tonsillar infection. In such cases, the removal of the tonsils very often clears up the systemic condition and the patient is considerably improved in every way. It is therefore surprising to note in the cases about to be related that the tonsils had been removed in some of them that were the most serious. This may be accounted for by the fact that an increase in lymphatic tissue frequently takes place in the mucous membranes of the oropharynx after the removal of the tonsils and that such tissue may become infected and cause a very serious condition. Again, there are cases in which a fairly good tonsillectomy has been performed and yet a small piece of tonsillar tissue remains, sometimes no larger than the head of a pin. For example, Mrs. S. was crippled with arthritis and was bed-ridden for six months. Her physician, a most competent man, insisted that

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there was a small infected area in the left tonsillar fossa. Examination showed that the tonsils had been well removed and it was impossible while examining her in bed for me to see such an area. However, she was able to come to my office some few weeks later, at which time a tiny drop of pus could be exuded from the minute mass of tissue no larger than the head of a pin. This was removed a few days later, after which the rheumatic manifestations entirely disappeared.

We shall eliminate from this treatise all mention of such conditions as syphilis, ordinary Vincent's infection and diphtheria and confine ourselves to four conditions which have seriously come to my attention during the past three years.

1. *Parapharyngeal Abscesses:* Cases of this type frequently resemble the ordinary peritonsillar abscess. The throat is considerably swollen on one side, the anterior pillar of the fauces bulges considerably and there is every evidence that a large amount of pus is present. Such a condition may occur even after the tonsils have been removed and the exact location of the abscess is problematical. One is often surprised on plunging into the cavity with a sharp pointed forceps and exploring it under general anesthesia, to find no pus. As a rule this means that a small amount of pus is present but is so admixed with the free hemorrhage that it can not be seen. I shall cite two cases.

a. Some years ago a little girl of 10 developed an acute infection in the left side of her throat. Her tonsils had been well removed. On the fourth day the swelling had so increased in size and the discomfort was so great that it was decided to open up the possible abscess. Another laryngologist had charge of the case. He plunged a knife into the cavity, whereupon there was an expulsion of a small amount of pus. The patient was considerably relieved and after four days was allowed to get out of bed. On this fourth day she was given a piece of hard candy. Suddenly, when biting on it, there was a profuse flow of blood from the throat and the patient went into immediate collapse. The hemorrhage could not be stopped for some time until someone exerted pressure on the carotid artery. She was given a fair dose of morphin and allowed to sleep and after a few hours a transfusion of 500 c.c. of blood brought her around. Examination of the throat at that time showed no evidence of the recent hemorrhage. A short time after the transfusion, a facial paralysis of the same side and a complete hemiplegia of the opposite side occurred. This paralysis, to a certain extent, remained permanent and the young woman today is walking around with a decided limp and a partial

droop to her face. The cause of the hemorrhage is problematical. It is my opinion that a branch of the internal maxillary artery was ruptured in the abscess cavity. After the hemorrhage ceased, a clot took place in an aberrant vessel which went through a dehiscence of bone in the skull which definitely shut off the blood supply from a certain part of the brain.

b. A little boy of nine had been suffering from some severe throat infection, which eventually resulted in the formation of a large swelling around the left tonsil. He had been under the care of his family physician, who called me in consultation. Examination showed a typical peritonsillar swelling. Plunging a sharp pointed forceps into the cavity, I was surprised at the unusual hemorrhage and small amount of pus. I sincerely expected the condition to clear up but, on visiting him a few days later, I found the left tonsil extruding into the throat with a large palpitating area behind it. I advised that the patient be removed to the hospital at once and that the tonsil be enucleated under general anesthesia. The operation was extremely simple. In fact the tonsil had become so loosened that it could almost be swept out of the throat with the finger. Exploration of the diseased area behind it proved that we had an edematous mass of tissue which no doubt would subside now that the original source of infection had been removed. Above it was a small sinus, which was probed. The probe sunk into a cavity for about 1½ inches and revealed no evidence necessitating further manipulation. The patient was brought to his room and the usual cleansing treatment to the throat given. Suddenly, 24 hours after the operation, there was a terrific explosion of a large amount of blood from his mouth and within one minute the patient died.

2. Cellulitis: A second type of case is that in which a severe throat infection takes place oftentimes after the tonsils have been removed. They evidence themselves mainly by considerable swelling and edema of the pharyngeal tissues on one side behind the posterior pillar of the fauces and are definitely a cellulitis. Of course, one suspects pus and so numerous incisions are frequently made into the bulging area and one is often surprised to find no pus. However, such incisions, with drainage of area involved, will result in a complete resolution of the symptoms in most cases. One may liken such a condition to an infection of the finger, minute in itself but of sufficient virulence to cause a severe cellulitis of the entire arm. In such an infection, the surgeon not only opens the original infected area but frequently makes numerous incisions along the arm. One knows the seriousness

of such an infection; it may be equally as serious when it appears in the throat.

In spite of the fact that I have seen quite a number of cases of cellulitis of the pharyngeal tissues, I have been able to relieve every one of them with a single exception.

a. Miss R., age 20 years, consulted me in my office for a severe soreness and pain on the right side of her throat. Examination showed a slight boggy swelling of the pharyngeal tissues extending up to the nasopharynx and down toward the larynx. The patient was not sick enough to be put to bed and came to the office the following day. At this time the swelling had extended to the center line but did not seem of serious importance. However, I advised her to go to the hospital and to be under observation. As she had some difficulty in swallowing and her temperature had gone up, I decided to explore the swollen area. Numerous incisions, under general anesthesia, revealed no pus. During the course of the next few days the throat condition subsided considerably and eventually cleared up. However, the temperature continued to rise and the patient looked extremely ill. A swelling was then noticed over the right thyroid lobe, which retrogressed under ice applications, only to be followed by a similar swelling on the opposite side. Gradually the patient noticed difficulty in swallowing. She developed a slight cough, had increased respirations, and consultation with other men made me feel that she had developed a broncho-pneumonia. X-rays seemed to confirm this opinion. Observation over a period of two weeks showed a continuance in the temperature, extreme prostration and a high blood count. At the end of the third week a thoracic surgeon was called in consultation, who suggested an infection of the mediastinum. A long needle was plunged into the mediastinum at about the eighth dorsal vertebra and a syringeful of straw-colored pus, in which fibrin was intermixed, was drawn out. The patient was immediately taken to the operating room and, under local anesthesia, two ribs were resected, the mediastinum opened. The cavity was filled with straw-colored fluid. It was divided into numerous smaller cavities by fibrinous deposits. The entire area was cleaned out and the patient returned to her room. The following day she expired.

This case should be a decided lesson to all of us. Realizing the lymphatic drainage of the mucous membranes of the throat and of the neck, and that such drainage can extend lower into the mediastinum, one should be on the lookout for such a complication. It probably occurs more often than one supposes.

3. Septic Sore Throats: The majority of physicians do not appreciate the seriousness of septic conditions of the throat which may

occur even in patients in whom the tonsils have been removed. The most serious cases are those in which the patient is extremely prostrated and in which there are numerous superficial ulcerations on the mucous membranes. These patients do not run a high temperature as a rule nor does the blood count reveal the seriousness of the condition. Within a period of three years I had three deaths from so-called septic sore throats. All of these patients had been receiving ambulatory treatments and were never seriously ill until a few days before they died. Another type of case is that in which the patient has never complained of any throat condition. He usually feels below par, runs a slight temperature, particularly at night, has chilly sensations and gradually loses weight. The average physician does not go to the trouble of taking a blood count in such cases and blood cultures are never thought of until the patient is in extremis. The following case is illustrative:

a. Mr. V. had been a patient of mine for the past 20 years. During the summer two years ago he began to feel under par and consulted his family physician. He had a slight temperature in the evening, frequently was chilly and had no appetite. He gradually lost weight. His family physician advised him to go into the woods for a few weeks, lead an active healthy outdoor life and then return. When he came back he was no better than he had been before. His wife thought that he might possibly have some sinus condition and therefore brought him in to see me nine weeks after the onset of his trouble. Examination of his nose and sinuses was negative but pressure on his tonsils showed them full of pus. These tonsils were small and buried. Culture of the pus showed a streptococcus hemolyticus. Patient's temperature at this time was 103° rectally. I advised him to go to the hospital at once and insisted upon immediate removal of the tonsils. The operation was comparatively easy. A culture of the pus was made and a vaccine prepared. I found on operation that the right tonsil was mainly infected with a large abscess cavity behind it and that the left tonsil was comparatively free from pus. The recovery from the operation on his throat was uneventful but the temperature did not subside and the patient did not improve. A blood culture taken two days after operation showed hundreds of colonies of streptococcus hemolyticus. Two transfusions were given which did not retard the infection. As most of the infection was in the right tonsil and as, apparently there was definite absorption from the deeper tissues in this region, I felt that we were very probably dealing with a septic condition of the veins in the tonsillar area. Reasoning from this premise, I felt that a resection of the jugular vein, after allowing of a free flow of blood from the upper end, simi-

lar to a procedure outlined for sinus thrombosis after mastoid operation, might result in some good. However, the patient continued to get worse. Numerous consultations were held with eminent authorities. No other focus of infection could be found. Later an endocarditis developed and, at the end of five weeks, the patient died.

4. Agranulocytic Angina: In this day one hears of a great many cases of agranulocytosis. I have recently published a paper on this subject, citing three cases. I do not wish to go into the details of all these cases here but one must realize the seriousness of such a condition and should be able to make a diagnosis at an early date. There is nothing typical about agranulocytic cases except the extreme prostration and certain evidences in the throat. Most of these patients in the beginning do not have any very serious complaint and therefore very little attention is paid to the throat condition. Gradually they become more prostrated and the throat presents a peculiar picture. The mucous membranes are dry, there is swelling and edema of the tissues in the tonsillar regions and often evidence of a bogginess which suggests a peritonsillar abscess. There is a peculiar odor exuding from the mouth which is hard to describe but is indicative of a gangrenous condition. The diagnosis is clinched by a blood examination which shows an extremely small number of white cells with a decided reduction in the number of polymorphonuclear cells. In one of my cases the white blood count was six hundred with one per cent of polymorphonuclear cells and in another case the white blood cells and the polymorphonuclear cells had disappeared entirely. Most of these patients die very rapidly. A certain number of them have been able to overcome their infection when it has been discovered in the early stages and definite medication given. It is often possible to be misled. For example, in one of the cases, the patient was brought to the hospital with a diagnosis of pneumonia. The white blood count was 25,000, with 90 per cent of polymorphonuclear cells. I saw the patient in an oxygen tent. She had a peculiar facies and I suggested to the doctor a possible agranulocytosis. A blood count taken the following day showed fifteen hundred white blood cells with two per cent of polymorphonuclears. The patient died two days later.

Although the manifestation in agranulocytosis is in the throat, one knows that there is a definite affection of the bone marrow. It is a question in my mind whether this infection is the primary source of the trouble or whether the throat manifestation comes first. However, the disease is of such serious import that one should always be on the lookout for it and realize that only under unusual circumstances do such patients fully recover.

Although it is possible that there are many other conditions of the throat which are baffling and serious, the cases cited above are of sufficient interest to make one appreciate that the examination of this area of mucous membrane should be a thorough one. It has impressed me that I have had so many deaths from throat infection in the past few years. We usually consider throat conditions of so little import that it is seldom that we acquaint the patient with the possible consequences until it is too late. Having had this experience, I insist that all patients suffering from so-called septic sore throats or suffering from any other throat condition that seems at all baffling, go to the hospital at once for careful observation.

64 East 58th Street.

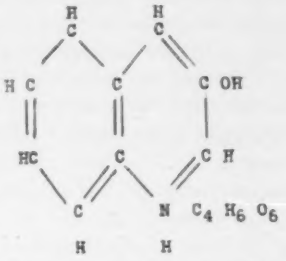
Barenberg and Lewis, of New York, in the Jan. 16, 1932, issue of the *Journal A. M. A.*, write on "The Relationship of Vitamin A to Respiratory Infections in Infants." The article is probably of added interest because of the tremendous advertising campaign by commercial houses to the general public on the value of vitamins in the fighting of winter colds. The authors discuss the literature and present, by means of tables, the results of some carefully carried out experiments. They reached the conclusion that there is no significant differences in the incidence or severity of respiratory infections because of the addition or lack of Vitamin A in foods.

Weiss, of Chicago, in the Feb. 13, 1932, issue of the *Journal A. M. A.*, presents a short article on "Primary Abscess of the Epiglottis." The pathology of this rare condition is described, following which the author presents a very interesting case of the condition. The symptoms were hoarseness, dysphagia, wheezing and tenderness. Indirect inspection revealed a large red swelling involving the lingual surface of the epiglottis with two pea-sized white areas of localized necrosis. The interior of the larynx was hidden from view. When the epiglottis was drawn forward with a curved probe, a portion of the vocal cords was seen to be normal. The author treated this case with incision of the swollen areas and followed the case to complete recovery in the course of three or four days.

A NEW ORGANIC COMPOUND FOR USE IN CHRONIC EAR SUPPURATION.*

DR. SIMON STEIN, New York.

This is a preliminary report on the use of oxy quinolin tartrate in forty-nine cases of chronic suppurative otitis media. This new chemical compound, which has proven to be a very powerful and efficient bactericidal agent, is the result of the work of Dr. Theodore Schlauch at the Midtown Hospital. The chemical formula is:



It is formed by the combination of a tartaric acid radical with one of the nitrogen ions and has the nature of a tertiary amin. It is alkaline, nonirritating, noncoagulating and highly germicidal. It is put up in the form of effervescent tablets, which allow the slow spread of the germicidal agent throughout the middle ear area. Due to its alkaline reaction, the mucus capsule of the pus is broken up, allowing the germicide to penetrate the bacterial discharge. There is no pain or irritation of any sort.

Every otologist encounters a number of chronic purulent middle ears which do not respond to the established methods of treatment. One can readily understand that this would follow because of the variety of etiological factors and complicating pathological conditions which are present in the individual cases. Some are due to a particularly resistant form of bacterial organism, and the others to a number of local factors which keep up the process, such as: *1.* infected granulation tissue; *2.* necrotic bone; *3.* open Eustachian tubes with *a.* infected tonsils and adenoids, and *b.* nasal sinus infection.

The treatments for these suppurative ears are many, and usually include irrigation with the use of different types of bactericidal agents, like the mercurial and the dyes. The following case reports are illustrative of the ears treated:

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*Presented at the October Meeting of the Midtown Hospital.

Case 1: J. F., age 7 years. *Physical Examination:* Ears: ROMPC entire drum destroyed, filled in by granulation tissue, profuse odoriferous discharge. Nose: Purulent discharge in both infranasal fossa. Throat: Diseased tonsils and adenoids. X-ray: Clouding of both antral sinuses.

Procedure: The tonsils and adenoids were removed and the antral sinuses were punctured and opened wide for proper drainage.

Irrigation of the ear, which had been kept up for several years, was stopped. The discharge was cleaned out by suction and then 25 per cent silver nitrate applied to the granulation tissue; following this a tablet was placed in the cleansed ear canal up against the granulations. This same treatment was followed out every third day and after the second treatment there was a material lessening both of the discharge and the extreme odor. This particular case required eight treatments. There were eight other cases of this type and the number of treatments required to clear them up varied from eight to twenty. The ears were perfectly dry and the granulation tissue was replaced by a firm fibrous layer.

Case 2: G. M., age 29 years. Radical mastoid operation was performed six years before. There was still a persistent odoriferous discharge.

Physical Examination: Ears: ROMPC. A large amount of granulation tissue with a thick odorous discharge. Nose: Negative. Throat: Diseased tonsils.

Procedure: The tonsils were removed and the discharge cleaned out by suction. Silver nitrate, 25 per cent, was applied to the granulation tissue, and a tablet inserted. This patient was given twelve treatments, one every third day, after which the discharge completely stopped, and there was a clean fibrous area left.

Case 3: C. R., age 22 years. This patient had a discharging left ear for about eighteen years, following scarlet fever in childhood. A radical mastoidectomy was performed on this ear, but there remained a foul persistent discharge.

Physical Examination: Ears: LOMPC. The external ear canal was filled up with granulation tissue and with a foul purulent discharge. Nose: Obstructive deflected septum to the right. Purulent discharge bilateral in the infranasal fossae. Throat: Negative. X-ray: Sinuses showed clouding of both antrae.

Procedure: Submucous resection and the antrae were opened wide for drainage. Locally, suction; silver nitrate to the granulation tissue and the tablets placed *in situ.* Intranasal treatment for the sinuses was also included. By the end of three months the ear was perfectly dry and healed over. There were five cases of this type.

Case 4: B. R., age 17 years. Discharge from the left ear for the past three years, with frequent attacks of tonsillitis.

Physical Examination: Ears: LOMPC; small central perforations of the drum; profuse pus discharge; no granulations. Nose: Negative. Throat: Hypertrophied diseased tonsils.

Procedure: Local tonsillectomy. The ear was treated by suction and by the insertion of a tablet against the perforation of the drum. This case was seen every third day for seven treatments. At the end of this time the perforation had closed over and there was no discharge. There were eight cases of this type.

Case 5: E. K., age 29 years. Discharge from both ears for the past three years.

Physical Examination: Ears: Large central perforations filled in with granulation tissue. Heavy pus discharge. Nose: Negative. Throat: Negative.

Procedure: Twenty-five per cent silver nitrate applications to the granulation tissue after cleansing by suction, followed by the insertion of the tablets. This case cleared up in four months of treatment. There were thirteen cases of this type.

Case 6: G. H., age 24 years. Has had a discharging right ear for the past two-and-one-half years.

Physical Examination: Ears: ROMPC; large marginal perforation with foul discharge. Nose: Negative. Throat: Negative.

Procedure: Suction and the use of the tablet; ordinary cleansing of the nose and throat. This patient has been under treatment for six months and though the discharge has lessened considerably, there still is some odor. The patient is a good deal more comfortable, but I believe that a radical mastoid will have to be performed in order to clear this case up entirely. There were five cases of this type.

Case 7: E. A., age 17 years. Had a discharging right ear for seven years.

Physical Examination: Ears: ROMPC; small central perforation in the drum, with a mucopus discharge. Nose: Catarrhal rhinitis. Throat: Negative.

Procedure: Suction and the application of a tablet. This patient had been under treatment for five months, but there still is a scanty mucus discharge, which becomes more profuse whenever the patient sets up an acute rhinitis. He is perfectly comfortable and believes that he has no discharge, because of the slight amount present. This condition is due to a patent Eustachian tube, which reinfects the middle ear constantly, making it extremely difficult to clear up. There were three cases of this type.

Case 8: C. B., age 35 years. Had a persistent ear discharge since childhood. This discharge was foul smelling and varied in amount at different times. The nose was always blocked up.

Physical Examination: Ears: ROMPC bilateral; drums entirely gone; filled in by granulation tissue; profuse discharge. Nose: High septal deviation to the right, heavy discharge, large ethmoidal polyps. Throat: Chronic pharyngitis. X-ray: Clouding of all the sinuses.

Procedure: High submucous resection, removal of the polyps, with drainage of all the sinuses. This was followed by the local use of silver nitrate to the granulations of the middle ear, and the use of the tablets. This patient cleared up in three months' time. There were four cases of this type.

Case 9 (Case of Dr. P. Kaufman): Pain and discharge from the right ear for two years.

Physical Examination: Ears: Right external otitis and eczema. Nose: Negative. Throat: Negative.

Procedure: Cleansed the ear by suction and placed a tablet in the canal. This was continued for three treatments, after which time there was an abeyance of the symptoms. Previous to this all sorts of remedial measures were tried, including X-ray exposures, without any relief.

Of the cases included in this report, forty were private cases of my own, the other nine were those of my associates, Dr. Hershkowitz and Dr. Kaufman, whose results coincided with my own.

DISCUSSION.

The cases enumerated are representative of various modifications of one and the same condition; namely, a chronic discharging ear, due to a necrotic and granulating area in the middle ear, in which the infection persists as a result of an accompanying intranasal sinus infection or diseased tonsils. The treatment of necessity is:

1. Eradicating the complicating factors, *e. g.*, sinus infection, diseased tonsils, or local bone necrosis. *2.* Removal of the granulation tissue. *3.* The use of a powerful germicide which will penetrate the crevices and angles of the middle ear.

The pus found in the middle ear usually has a good deal of tenacious mucus, which surrounds the bacteria, preventing the action of the germicide used. This compound is alkaline in reaction and dissolves this mucus capsule. The area involved in the middle ear is very sensitive and the average germicidal agent used is either very painful or irritating to the patient, and may produce an eczema of the canal. This compound, however, is nonirritating and painless. A number of chemicals which are used in infections of the ear have a coagulating effect on the surface of the tissue they come in con-

tact with, and thus prevent penetration. This compound does not coagulate. A number of chemicals put up in the form of a powder and blown into the middle ear have, first, an intensely irritating effect, especially in those patients with an open Eustachian tube; and, secondly, they cake and form a mass, which blocks the discharge and prevents proper reaction. This compound put up in the form of a tablet is easily introduced into the ear and effervesces slowly, giving no irritation, and spreads into every point where the bacteria may be. It leaves no residue and does not interfere with the egress of any discharge. We believe that we have in this chemical what is up to the present time the ideal germicidal agent for chronic ear discharge. Its simplicity of application, coupled with the absolutely nonirritating effect on the patient, and its extremely powerful germicidal effect, should appeal to all otolaryngologists who meet these chronic persistent middle ear infections, which fail to respond to the average form of treatment.

The procedure in brief is as follows:

1. Elimination of all neighboring infective areas, *e. g.*, the nose and the throat, and local bone necrosis which may require radical mastoidectomy.

2. Stop all irrigations; clean out the ear canal by gentle suction through a long, narrow metal tube, the tip of which can be carefully manipulated under the eye of the operator so as to produce no trauma. When the tablet is inserted the slight moisture left starts its effervescence.

3. Silver nitrate, 25 per cent, is used to remove any granulations present by slow cauterization.

4. The tablet is inserted with a pair of bayonet forceps and placed against the middle ear area, the granulation tissue, or the perforation of the drum.

I have recently modified this technique somewhat by putting a drop of adrenalin into the ear just before placing the tablet *in situ*, with the idea of shrinking up the granulation tissue and the mucous membrane of the middle ear, to further enhance the action of this compound.

Most of the cases presented have had either infected sinuses or diseased tonsils and it may be rightly argued that any other form of treatment now in use, in conjunction with the elimination of neighboring infected areas, would clear up ear infections. That is true enough, but this method clears up a great many cases in which all the above measures have failed, is very simple, used with the least amount of annoyance to the patient, and gives results in less time.

I wish to dwell upon the fact that the use of this tablet does not result in 100 per cent cures. There are failures, but the proportion of results obtained are greater in number and much more satisfactory than those of any other method now in use. This product is being used in other parts of the body where infections of a similar character are resistant to treatment, with equally satisfactory reports.

CONCLUSIONS.

1. Chronic ear discharges as a rule are extremely resistant to treatment.

2. There are a number of complicating infective areas which must be eliminated in order to clear up the discharge.

3. This compound is a powerful antiseptic which is nonirritating, noncoagulating, and one which can be used with the least amount of disturbance to the patient.

4. The cases reported were treated by various other methods, before this treatment was instituted.

5. This tablet in itself will not cure all chronic ear infections, but should be used by the otolaryngologist as another agent in his therapeutic armamentarium.

I wish here to express my appreciation to Dr. Sam Goldstein for his helpful criticism and to Dr. Theodore Schlauch for the generous way in which he supplied the tablets for experimental purposes. These tablets are now being used in acute middle ear infections, in nasal sinus and other nasal infections, and will be reported on at a later date.

STATISTICAL RESUME.

No. of Cases	*Granulations*	*Ear Drum*	*Complicating Factors*	*Result*
9	Large amt.	Absent	Diseased T & A antral sinusitis	Cured
1	Large amt.	Absent	Diseased tonsils	Cured
5	Large amt.	Absent	Septal obstruction antral sinusitis	Cured
8	None	Present, small central perforation	Diseased tonsils	Cured
13	None	Present, large central perforation	None	Cured
5	Small amt.	Present, large marginal perforation	None	Discharge less needs radical
3	None	Present, small central perforation	Catarrhal rhinitis catarrhal pharyngitis	Discharge less open tubes
4	Large amt.	Absent	Septal obstruction ethmoidal polyps	Cured
1	None	No perforation	Ext. otitis, eczema	Cured

225 East 19th Street.

International Digest of Current Otolaryngology.

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Announcement has been made that Prof. Dr. Erich Ruttin, of Vienna, will make a lecture tour of America in the near future. This will present to American otologists an opportunity to study under one of the foremost Viennese teachers.

Prof. Ruttin is a famous pupil of the famous teacher, Politzer. His long association with Politzer has trained him in the ways of the "father of otology." Prof. Ruttin is a shrewd clinician and in addition is one of the foremost pathologists of the world. This pathological experience has given him the background for his clinical deductions.

His labyrinthian work is well known and respected. During the World War he was in charge of a corps studying labyrinthian reaction in regard to aviation.

Those who have studied under Prof. Ruttin are most enthusiastic about his method of presenting labyrinthian reactions in his own peculiar, uncomplicated manner. His well modulated English, his clear deductions and his wonderful personality make these complex reactions seem surprisingly simple.

Prof. Ruttin's lecture course is as follows: March 21-26, Cincinnati; March 28-April 2, Kansas City; April 4-8, Des Moines; April 11-15, University of Kansas; April 18-22, Oklahoma City; April 25-29, Memphis; May 2-6, St. Louis; May 9-13, Madison, Wisc.

American otologists are strongly recommended to take advantage of hearing Prof. Ruttin. Further information regarding the course may be obtained by addressing Mr. Harold H. Ingham, University of Kansas, Lawrence, Kan.

Notice has been received that the Revue de Laryngologie, Otologie and Rhinologie is now being published by Georges Portmann, of Bordeaux, and edited by Delmas, of Bordeaux. The Revue is now

in its fifty-third year, having been established by E. J. Moure, in 1879.

The new policy of the journal will be to run forty pages each issue of the Review of Current Literature; it will also include transactions of scientific societies.

Fowler, of New York, in the Jan. 16, 1932, *Journal A. M. A.*, presents an article on "Clinical Research in Otolaryngology." He points out that the specialty is just entering a new era in otolaryngology in which the future looks brighter than it ever has before. He derides the specialist who makes the statement that our specialty has reached its limit or that there has been no real advancement in surgery except in the technique of execution.

Fowler believes that every clinician is able to carry on clinic research in his every-day clinic and private practice. The first step in this research is to maintain adequate records. Proper charts, properly recorded over a period of time, will disclose much information and will show the way for further research on subjects that we today pass over with a feeling that there is nothing more to do.

Looper, of Baltimore, in the Oct. 31, 1931, *Journal A. M. A.*, has an article on "Bronchoscopy as an Aid in the Diagnosis of Obscure Pulmonary Diseases." The purpose of the article is to emphasize the fact that foreign body work in bronchoscopy is only a small part of the useful work which may be done in competent hands.

The study of obscure pulmonary diseases may be greatly facilitated by means of bronchoscopy, and in numerous cases a diagnosis could only be reached by means of bronchoscopy when all other methods of investigation had failed.

Close co-operation between bronchoscopist, internist and surgeon is essential for good results.

Looper presents in detail fourteen interesting cases in which the bronchoscopist was of great service not only in establishing the diagnosis, but also in the treatment of the various conditions.

The article is concluded with the author's earnest hope that all large tuberculosis hospitals and sanataria will soon establish bronchoscopic clinics.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLARYNGOLOGY
AND THE
AMERICAN COLLEGE OF SURGEONS.

Meeting of Oct. 13, 1931.

The Activities of the Otological Research Laboratory of the Johns Hopkins University During the Past Five Years. Dr. S. J. Crowe.

The Otological Research Laboratory of the Johns Hopkins University was organized in 1924 for the purpose of studying the finer anatomy, the pathology and the physiology of the ear. The pathology and anatomy may be demonstrated with temporal bones collected at random in the autopsy room, but a study of the physiology of the human ear is a more complicated procedure. For example: No temporal bone is of value in this investigation unless an accurate test of the function of the cochlea and vestibular apparatus was made before death. The specimen must be obtained within a few hours after death. The preparation of good histological sections is so essential that approximately 1,000 temporal bones and several years of concentrated effort of the entire laboratory staff was required to develop a satisfactory technique for fixation, decalcification and embedding. Our plan from the first was to examine with the greatest possible accuracy the hearing of every hospital patient whose general condition makes it seem likely that he will come to autopsy. Many of these patients are found to have normal hearing. The histological sections of the temporal bones of such cases have been of great value, however, in developing a histological technique that is free from artifacts.

The testing of patients on all services throughout the hospital, the difficulties of securing the temporal bones of the tested cases and what once seemed an almost impossible task—the preparation of histological sections good enough to correlate with the functional tests—are all important steps in the development of this plan of investigation that are now satisfactorily solved. The most important and perhaps the most difficult task, however, that has not been solved, is the interpretation of the audiometer curve and tuning fork tests. It is absolutely essential that the otologist should be able to determine from the tuning fork tests, the audiometer record and the general diagnostic study: *1.* whether the lesion is located in the middle ear, the inner ear or the auditory nerve; or *2.* whether there is a lesion in both the middle and inner ear. He must also be familiar with the common etiological factors of the various types of deafness. In

other words, the diseases of the ear should be studied with the same methods used so successfully in general medicine, *i. e.*, the correlation of clinical observations and functional tests with the pathological findings. A clear understanding of the cause and pathology of hearing defects is absolutely essential. A clear understanding of the physiology of the various structures of the middle and inner ear, however, must come first.

The brilliant work of Wever and Bray has given us a method for studying in experimental animals the part played by each separate structure in the middle ear in the transmission of sound. Every nerve impulse is associated with a change of potential in the nerve trunk. Sound waves are transmitted by the middle ear structures and the perilymph and endolymph of the inner ear to the organ of Corti, where the mechanical stimulus is changed into nerve impulses. The auditory nerve of an animal under general anesthesia is exposed. One electrode is placed on the nerve and another in the muscles of the animal's neck. Wires lead from these electrodes to an amplifier in another part of the building. A loud speaker or telephone receiver is connected with the output of the amplifier. If the animal's ear is normal, spoken words and the tones of tuning forks (from 128 d. v. to approximately 5,000 d. v.) entering the ear are reproduced in the loud speaker with clarity and distinctness. The middle ear of a cat or a dog may be exposed through an incision in the neck without injury to the drum or ossicles. This method makes it possible to demonstrate in an experimental animal the relative importance of the tympanic membrane, the ossicles, the tensor tympani and stapedius muscles and the round window membrane in the transmission of the spoken voice or the various octaves in the tone scale mentioned above. A detailed description of the experiments has been published so only the most important conclusions will be given here: *1.* The clearness and resonance of the voice is diminished by any change in the length and diameter of the external auditory canal; *2.* pure lesions of the drum that interfere in no way with the ligaments or movements of the ossicles have very little effect on the transmission of words or tuning fork tones; *3.* any experimental lesion that tends to interfere with the movements of the ossicles or *increases* the rigidity of the ossicular chain causes a marked impairment in the transmission of *low* tones; *4.* division of the tensor tympani muscle, which *decreases* the rigidity of the ossicular chain, causes a marked impairment in the transmission of *high* tones; *5.* puncture of the round window membrane, which allows a drop of the perilymphatic fluid to escape, results in a *profound* loss in the transmission of all sounds;

6. pressure on the round window membrane, which makes it tense and reduces its mobility, increases the sensitivity of the cochlear end organs and the clearness and intensity of words and tuning fork tones are *increased* approximately 50 per cent.

These facts could never be demonstrated in a patient or in histological sections of the human ear, because infection usually involves to a greater or less degree all of the middle ear structures. Only a beginning has been made in these studies but it is evident that the method will enable us to gain a clearer understanding of the function of each separate structure in the ear and the type and degree of impairment of sound transmission that results from a lesion of one or more of these structures. This type of experimental work, together with the functional, histological and statistical studies on patients, will in time enable us to intelligently interpret the history, the general physical examination and laboratory tests. The local findings in the ear and upper air passages, the various tuning fork and audiometer tests thus make it possible to locate the lesion and understand its cause. When this has been accomplished we may then hope to arrest the progress of deafness or possibly prevent its development.

After this brief statement of the activities of the laboratory, the remaining time was taken up with a demonstration to show how impossible it is to differentiate, *with the audiometer curve alone*, between pure conductive lesions in the middle ear, pure cochlear lesions due to a degeneration of the cochlear end organ or cells of the cochlear ganglion, or a pure lesion of the cochlear nerve in the internal auditory canal or brain stem. The audiometer curve is of little value in the localization of the lesion unless it is supplemented with accurate tuning fork tests.

A series of 100 lantern slides of "tested cases" were shown. First, a series of normal audiometer charts were shown, with sections from the middle and inner ear and nerve showing normal anatomical structure. Then cases with a pure middle ear lesion, cases with a pure cochlear lesion, cases with a pure nerve lesion and, finally, a series in which the impairment of hearing was due to otosclerosis. These were all autopsy cases whose hearing had been carefully tested. Following each audiometer chart a series of slides from the middle and inner ear showed the exact location and type of lesion, or combination of lesions in each case.

One great defect in this type of investigation is the necessity of supplementing the purely *objective* audiometer test for the perception of speech and pure tones with the *subjective* tuning fork tests of bone conduction time.

DISCUSSION.

DR. FOWLER: It is probable that I was invited to discuss this paper because I have been to Baltimore and am familiar with a good deal of the work being carried on there. There is no need for me to praise the research work at Johns Hopkins. It speaks for itself, and I shall not take further time in eulogy.

Throughout this evening's presentation one thought has persisted in my mind, *i. e.*, pathological research will accomplish but little unless it is co-ordinated with clinical observations. Pathological findings, whereas they show the conditions shortly before or following death, necessarily do not show the conditions which precede death any considerable length of time. For instance, take our audiometric tests of the hearing capacity in patients who we have reason to believe will soon die. We cannot examine these patients immediately before death and the great majority of them tend to live for weeks or months after we have examined them, so that our findings at autopsy do not show the conditions which existed in the ear at the time of our examination. Of course, this cannot be helped, but, even so, microscopic sections will show the lesions causing deafness plus the lesions caused by the terminal pneumonias or other terminal infections. It is therefore important that we do not slight our clinical observations, and in fact there is no clinic so small or no practice so limited that every laryngologist cannot institute upon a small scale at least clinical research. In the end, of course, it is the clinical test which alone is convincing, insofar as remedial measures are concerned.

I have jotted down a few points in Dr. Crowe's address to which I will allude. There is no such thing as normal hearing because people in their hearing differ as much as twenty units up or down from one another, without their hearing being necessarily abnormal, as far as they individually are concerned. Therefore, there is a normal hearing only for each person individually. This consists in the most perfect hearing, the particular healthy ear can attain. Collectively we can refer only to average normal hearing. It follows, therefore, that in some of the charts that Dr. Crowe has shown with lesions in the middle ear coincident with a diminution in hearing of only 10 or 15 S.U., it may be that this hearing is far from normal, although compared to a low average normal standard it appears almost perfect.

I would point out that it is almost impossible to have a single lesion causing deafness. Anything that nature does to the middle ear in disease, or anything we do experimentally, has many effects besides the obvious primary effect. But on the whole the classical interpretation of the tests appears quite logical if we will take into consideration the various factors producing the abnormal hearing.

The mere presence of adhesions as seen microscopically does not necessarily mean that these adhesions are producing as much effect as we imagine. They may be very soft, loose adhesions, they may be very dense, hard adhesions. The loose adhesions will produce mainly a loading effect, the dense adhesions, mainly an immobilizing of increased tension effect. Loading of the transmitting mechanism (as I showed many years ago by placing a lead shot on the tympanic membrane) causes diminution of hearing for the high tones unless the loading is so situated as to increase the tension of the drum and ossicles, in which case the high tones being favored by an increase in tension may be heard better than could have been expected under the conditions. In old people we very regularly find a rapid progressive loss as the high tone scale is ascended, but in my experience this does not necessarily mean a syphilitic nerve deafness. As a matter of fact, it may be present from nerve deafness in a patient who has syphilis, and still show at autopsy no syphilitic lesion affecting the nerve or other parts of the apparatus of hearing.

Narrow, abrupt deficiency loops in the hearing are, it is true, usually caused by a lesion in the cochlea most often probably involving the spiral ganglia cells, but it must not be forgotten that tinnitus (often of the frequencies included in the deficient area) by its masking effect, may cause or accentuate these limited deafened areas. This is proven by the fact that when the tinnitus stops the hearing may improve as much as 30 to 40 S.U., although some local

disturbance persists, as is evidenced by a failure of the hearing to return quite to the level found on either side (or brink) of the dip.

Otosclerosis Findings: We also have discovered incidences where there is no deafness in the presence of otosclerosis, which makes one believe that, after all, otosclerosis is hardly a disease; unless it happens to progress to the point where it interferes with the motion of the footplate or stapes. There is no doubt that otosclerosis has a starting point, a progressive phase and a stopping point. The thing we have to look for is not the removal of the bone (that cannot be done)—but if we can get at the cause of the start and of the continuation and of the stop of the growth (or of any of these) we will be able to in some measure control deafness caused by otosclerosis. This is one reason why in clinical work we should make audiometer tests, and careful examinations by blood chemistry, blood count, X-rays, etc., in at least selected cases. I believe that by doing these things and eventually comparing the results we will find out where, why and how things start and continue or stop. You may be wondering something of the same sort about me, so it may be well to end my discussion at this point.

DR. S. J. CROWE: I am glad that Dr. Fowler emphasized the possibility of the general medical profession being able to help in an investigation of this kind. If enough men throughout the country interest themselves in making careful functional records and then secure the temporal bones and forward them to the laboratory in New York or Baltimore, it would add greatly to the amount of material available for study.

Some Intimate Studies of Nasal Function; Their Bearing on Diagnosis and Treatment. Dr. A. W. Proetz.

(To be published in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. F. H. PIKE: One of the first things I was told about physiology was that it was the handmaiden of medicine, and I was told that in such a way as to understand that handmaidens should be beseeming in their conduct. It was implied that the duty of the handmaiden was to concern herself with the business of medicine. Physiology is apt to follow certain fashions; someone gets an idea and tries it out with certain apparatus; that idea appears to be a good one and eventually the research apparatus becomes an affair of highly polished wood, satin-finished metal and replendent chromium. The thinking was all done before the plans were sent to the factor; we are going on with something that is fashionable.

I was also told that there is such a thing as medical physiology. The reason for that will come out later. The handmaiden was supposed to busy herself with her mistress—look after medicine; but this shameless creature has been considering other things: she has consorted with oysters where cilia have been moving about, and she has even watched some of the ciliated protozoans under the microscope. One cannot see much about the action of the cilia as the organisms go across the field of the microscope at high speed. We used to add some gummy substance to the water to slow down the motion of the cilia. In more recent times we can out them on a slow motion picture film. I think that perhaps, after all, this handmaiden can give some account of herself on her Thursday afternoons out, when she is not considering medical physiology as such.

I don't know very much about cilia. I am not sure that anyone does. One of the best articles that I know of is that in Richet's Dictionaire de Physiologie, published twenty-five or thirty years ago; and there has been very little work on cilia since that time. The physiologists work certain things out, perhaps to their own satisfaction and with the apparatus available at that time, and the fashions change later on.

A similar situation arose in the case of the sympathetic nervous system. Gaskell and Langley, thirty or forty years ago, did work on the sympathetic system. Recently surgeons have done more work in that line. The physiologists have done little more until very recently, and the sympathetic system has remained very nearly where Gaskell and Langley had left it. So it happens

that the work on physiology and medicine is often not synchronized. Someone has curiosity to stray off into perhaps unfrequented or even forbidden fields to look at something which catches his fancy, and it may be a generation later before that becomes of importance in clinical medicine.

I never thought when I was watching cilia that ciliary action might become important in the treatment of sinuses, yet tonight it has been shown to be of importance. One contention we physiologists might raise is this: I am old enough to remember a lot of things that were used as nasal douches—some of them pretty strong. I remember a gargle of carbolic acid mixed with glycerin; in some remote way it was supposed to be harmless and still have the power of killing bacteria. That has always been a mystery to me; but we know that the ciliated protozoans are rather sensitive to some of the changes in their environment. In the human nose it seems a little different. We have the dust of the city to contend with, the vapor of unburned or incompletely burned gasoline, smoke and all the other floating matter of the air to contend with, and yet the action of the cilia is supposed or expected to continue. These cilia in the nasal passages amaze one by their resistance to adverse influences.

I have been speaking of ciliated protozoans that do not have any nerves at all; and in plants we have pretty good evidence that ciliary action can occur without any nerve action. One might suppose that sympathetic nervous action would be directed to maintaining circulatory and perhaps glandular conditions rather than being concerned directly in maintaining ciliary activity. The whole subject seems to be coming within the range of experimental study now. The physiologist is supposed to be omniscient. He is not. Way down in the bottom of his heart he will usually tell you there are some things he does not know.

The aim of physiology is, in general, to make some levy on the fundamental fields of science and get this knowledge together, even in an incomplete form, in such a way as to bear on some part of human tissue or human economy. It has certain methods of its own, and while no physiologist may work out all the facts, yet somewhere there is or should be a fund of fundamental knowledge and theory, and methods, which will enable others to go on with the study of these special fields. The methods and fund of fundamental knowledge available in physiology is likely to be large and varies in proportion to the amount of wandering one physiologist or another has done from the beaten or the fashionable path into more remote and seemingly unrelated or unprofitable regions.

We know that solutions containing all the inorganic solids will sustain ciliary action much longer in some ciliated infusions than will a solution of any one inorganic salt alone. That may offer a hint for some of our nasal treatments. It is one thing that might be worth trying. On the clinical side, I don't know. I am a physiologist, not a clinician.